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# THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—14TH YEAR. SYDNEY: SATURDAY, APRIL 23, 1927. No. 17.

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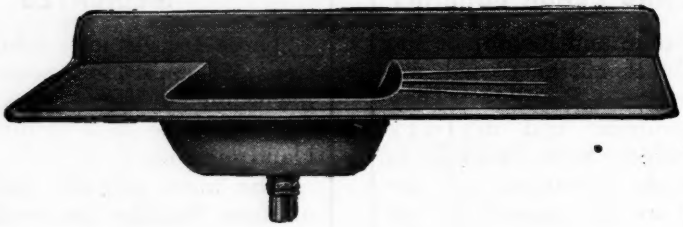

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# ANÆSTHESIA AND THE NEWER ANÆSTHETICS: ETHER.[1]

By Reginald Howden, M.B., B.S. (Melbourne),
*Honorary Anæsthetist, The Melbourne Hospital, Melbourne.*

It is quite impossible to say at what period in the world's history attempts were first made to prevent or relieve physical pain. The ancient Egyptians, Assyrians and Chinese were familiar with many vegetable substances capable of producing pleasurable, sedative and anodyne effects, but it was not until the very close of the eighteenth century that our modern system of anæsthesia began to be foreshadowed. The discovery of nitrous oxide by Priestley in 1774 marked the commencement of a new era in chemical physics and paved the way for the introduction of a system of inducing complete unconsciousness which has gradually developed into the art of surgical anæsthesia such as we have today. It is only a little over a century ago that an article appeared in an English journal believed to have been written by Faraday which states that when the vapour of ether mixed with common air is inhaled, it produces effects very similar to those occasioned by nitrous oxide. It was not, however, until 1846 that it was recognized that the inhalation of ether vapour could produce complete surgical anæsthesia. The next year Simpson introduced chloroform and in the same year ethyl chloride was first administered for a surgical operation. As my remarks are to be confined to the value of ether as a general anæsthetic agent, I shall do no more than refer only to the various theories which exist as to the mode of action of anæsthetics, the theory of lipoid solubility, theory of asphyxia, the precipitation theory *et cetera*. The hypothesis that anæsthetics act by decreasing the permeability of cells appears to be the most satisfactory explanation of the facts of anæsthesia. It must be remembered that anæsthetics are general protoplasmic poisons and will narcotize or kill all forms of cells if given in sufficient concentration. Nervous tissues are paralysed by lower concentrations of anæsthetics than are other tissues of the body and it is this fact which renders the use of anæsthetics possible.

There are degrees or stages of anæsthesia conveniently divided into four, analgesia, light anæsthesia or the excitement stage, deep anæsthesia or full surgical anæsthesia and bulbar paralysis. These stages vary with different anæsthetics and they can be followed by the progressive loss of reflexes. The power of voluntary movement usually disappears shortly before sensation and consciousness completely disappear. In the second stage the conjunctival and cough and vomiting reflexes disappear. In the third stage general muscular relaxation occurs and late in this stage the reaction of the pupil to light disappears. The fourth stage is that of paralysis with depression of the respiratory centre which, left unrecognized and untreated, finally produces death. The maintenance of an efficient ventilation of the lungs, that is a free airway, is the first essential in giving an anæsthetic. Depression of respiration causes a rise in carbon dioxide tension and in slight amount does not produce any serious injurious effects on the body, as it stimulates the respiratory centre and so tends to correct itself automatically. An oxygen tension decrease, however, is directly injurious to all the body tissues, the central nervous system and the heart being most rapidly affected. So the most dangerous derangements of respiration are those in which the oxygen tension is decreased, the carbon dioxide tension being raised. Respiration during the administration of anæsthetics may be interfered with in several ways.

(i.) The oxygen content may be reduced as by rebreathing methods.

(ii.) The anæsthetic may irritate the air passage and cause mechanical obstruction or later in the stage of narcosis, the muscles being paralysed, may produce obstruction.

(iii.) The respiratory centre may be depressed and may not be so sensitive to a rise in carbon dioxide.

(iv.) The anæsthetic may interfere with oxygen carrying powers of the blood. The anæsthetist by means of the respiratory sound judges the condition of the air passages. Snoring is the commonest of the adventitious sounds heard in respiration during anæsthesia and it must be looked upon as so much muscular spasm about the fauces which may be so pronounced as to cause an obstructive stertor and so endanger life. This is overcome by pressing forward the lower jaw and carrying the tongue with it and if this is not successful, by introducing a mouth gag and drawing the tongue forward. Should the obstruction still continue, an artificial airway such as a pharyngeal tube should be introduced which allows free passage of air to the pharynx. If still unsuccessful, the anæsthetist may pass a finger to the back of the throat and separate the base of the tongue and epiglottis from the pharynx, this approximation being a common cause of respiratory trouble experienced in edentulous patients.

This is quite a different condition to stridor which always indicates a tendency towards occlusion of the larynx itself. Minor degrees of stridor are overcome by stopping the anæsthetic, briskly rubbing the lips and pushing the lower jaw forward. If it continues in spite of treatment laryngotomy must be performed. Fortunately under ether anæsthesia this is not a common complication.

"Strained" respiratory sounds are generally of pathological significance. When there is strained respiration with considerable phonation, a corneal reflex being present, the cause is usually a collection of mucus in the larynx. Lightening the anæsthesia and allowing a cough reflex usually relieves it entirely. Moist respiratory sounds indicate the presence of fluid immediately above, within or below the larynx.

During induction the pulse rate is increased as the blood pressure is raised. This, however, is not

[1] Read at a meeting of the Victorian Branch of the British Medical Association on March 2, 1927.

maintained and in nearly all cases a fall occurs, the greatest fall being with chloroform and the least with gas and oxygen. The fall occurs earlier with chloroform than with ether, the fall with gas and oxygen being much later and on the cessation of the anæsthetic much more quickly restored.

During and after the administration of anæsthetics two forms of shock are encountered, a primary form, which is not of much importance, and is due to disturbance of the vasomotor centre and a secondary form, the chief clinical sign of which is a fall in blood pressure associated with pallor of the mucous membranes, small weak pulse and increasing rapid and shallow respiration. Operations involving extensive injuries to tissues, especially to muscle, are particularly liable to cause shock. The anæsthetist can help most in this by his choice of anæsthetic and by insuring a free air way, so that oxygen supply of the tissues is as good as possible. Heart stimulants are not of any use in this form of shock, intravenous injections of saline solution are rapid in action, but with 6% gum arabic added, the effect is much more prolonged and in desperate cases blood transfusion is more efficacious.

The administration of ether as a general anæsthetic may be (i) by the open method, (ii) by the closed or rebreathing method, (iii) by the rectal method, (iv) by the intratracheal method, (v) by the warmed ether vapour method.

As another speaker will deal with the intratracheal method of administration which has largely superseded the rectal method, I shall confine my remarks to the Clover closed method and the warmed ether vapour method. Ether acts as a slight irritant to the respiratory passages with consequent increased secretion of saliva and mucus and sometimes inflammatory after effects. It produces little action on the medullary vasomotor centres and it does not directly stimulate the heart, but is a feeble cardiac depressant.

By the open system is meant an administration so conducted that the patient inspires air laden with ether vapour from a mask covered with material which offers little resistance to inspiration and reduces rebreathing to a minimum. The material used varies with different individuals, it may be domette, flannel, lint or gauze. I generally use gauze of about six to eight thicknesses. Underneath the mask a pad of gamgee tissue or wool with an opening in the centre or a folded towel is placed and this acts in two ways. The whole of the respired air passes through the covering of the mask and any excess ether does not run on to the skin of the patient. The method of administration varies. Many prefer a preliminary injection of morphine and atropine, others give an injection of atropine and a dose of chloretone by the mouth, others again give an injection of scopolamine, atropine and morphine and in America preliminary medication with bromide and paraldehyde is recommended. Some prefer no preliminary treatment whatever.

Ether being an irritant vapour, many prefer using a less irritant vapour with which to start induction and then rapidly pass over to ether when sensation is blurred. That most generally used here is ethyl chloride and frequently chloroform. Some aromatic oil is used on occasions as a preliminary to the use of the two inducing agents or of ether itself. I find that most patients prefer ethyl chloride as the inducing agent, as it is rapid in action and not so unpleasant as chloroform or ether. When given slowly and with the mask raised and gradually lowered on to the face of the patient and with gradual increase of the concentration of the vapour it takes about four to six cubic centimetres of ethyl chloride for a female and six to eight cubic centimetres for a male patient before the change to ether can be satisfactorily made and most patients show or retain little objection to it. The time to change over from one of the inducing agents to ether is learned only by experience. In training students I generally advise them to ask the patients to count after them and so soon as they cease counting to change over. Some patients (especially the old soldiers) will refuse to do this and then I give ethyl chloride until stertor begins, when I make the change. Ether is steadily introduced into the mask and so long as respiration is free and regular, the mask is kept saturated. As anæsthesia becomes deeper the patient's head is turned on to one side so that the saliva which is secreted in fair amount, will collect in the hollow of the dependent cheek and not run back in the throat and irritate the larynx and obstruct respiration. The towel which has been used in the mask to concentrate the ethyl chloride is now unfolded and laid across the lower half of the mask, serving two purposes, to hold the mask in position and to increase the concentration of vapour. The mask is kept moist by occasional additions of ether, the depth of anæsthesia being gauged by the stertor and the pupil reflex. A finger of one hand holds the angle of the jaw and automatically keeps the jaw forward, if there is any indication of respiratory obstruction. As it is so general as to be taken almost as an axiom that respiration fails before circulation in ether administration, the student's attention is always directed to keep the air way free and if it is not free, to find out why and attempt to rectify it. Both amongst students and outside practitioners I think the tendency is to make the change over too late and to use too concentrated an ether vapour and also to neglect early signs of respiratory embarrassment or obstruction. It is not an uncommon sight to see a large patch of moisture on the pillow under the patient's head, due to excess ether running off the mask, a wasteful procedure. Use of the smallest amount of ether that will keep the patient in a state of full surgical anæsthesia is the ideal to be aimed at and this varies with every individual.

The eye signs are to be depended upon as an indication of the depth of anæsthesia. During the early stages the conjunctival reflex is pronounced, any attempt to lift the upper eyelid being strongly resisted. At a later stage this disappears and there is a roving eyeball, still later the eyeball becomes fixed with a small pupil, later the pupil dilates moderately and reacts briskly to light and this is

the stage which is usually most suitable for surgical anæsthesia.

One ear is always left exposed and the lobe of it is watched as a good indication of the state of the capillary circulation. Any blueness or pallor leads one to see first that respiration is free and second to watch for eye signs to see that anæsthesia is not too light or if pallor occurs late in the operation, to decide whether it is due to shock or to threatened vomiting and to treat it accordingly. If administration of ether is carried too far by overdosage, respiration begins to show signs of failure. The pupils dilate, the colour becomes dusky, the eyelids are separated and the pulse, though still good, is weaker and probably slower. In many cases the breathing loses its stertor, becomes feebler and then ceases. The all important point is that with ether administration, whenever respiratory failure occurs, the oxygen tension at the moment when breathing ceases is sufficiently satisfactory for remedial measures to be almost invariably successful, that is unless restorative measures, such as artificial respiration, are too long delayed, the heart is not likely to fail.

### Complications.

In early stages breath holding is often due to too strong vapour, but in other instances even with delicate vapour there is trouble and great patience is needed. In later stages shallow breathing may be troublesome; this can be combated by increasing rebreathing by folding a towel over the mask or covering it with the warmed hand, first using brisk lip friction. Early coughing is frequent in people with irritable throats and it may be necessary to use concentrated ether and bludgeon them through the second stage. Muscular rigidity is often due to concentrated vapour; a few breaths of air will often relieve or if there is respiratory obstruction, the introduction of a mouth gag drawing the tongue forward may relieve. It may be necessary to introduce a pharyngeal tube. Ether tremor is usually a sign of too light anæsthesia, but in some instances it can be stopped only by changing over to chloroform. Warmed ether is used by some anæsthetists by pumping a stream of air over ether and passing it through a coiled tube immersed in hot water or some other heating arrangement. The ether is carried to the patient by a tube inserted under the edge of the mask. Though theoretically this should result in a more satisfactory anæsthesia, it has not in my hands produced any better results than ether administered by the open method. Ether administered by the closed method of rebreathing such as by a Clover or Ormsby inhalater, with the exception of using less ether offers no advantages over ether given by the open method and has many disadvantages.

To sum up the method I advocate is a preliminary hypodermic injection of atropine, in a dose of 0·54 milligramme (one one hundred and twentieth of a grain) for males and 0·43 milligramme (one one hundred and fiftieth of a grain) for females and oral administration of chloretone 0·3 to 0·6 gramme (five to ten grains) half an hour before the anæsthetic is commenced. Induction should be by means of ethyl chloride or chloroform, followed by ether given by the open method in the manner described above. As mentioned before, the anæsthetic to be selected depends on the type of patient, the nature of the operation and the conditions under which it is to be performed and here I should like to say that the preparation of the patient is, I think, at times too drastic. The safest anæsthetic is nitrous oxide and oxygen and it is being increasingly used and chloroform is a much less safe anæsthetic than ether. But the safety or otherwise of an anæsthetic is very considerably effected by the experience or otherwise of the anæsthetist. Ether is also administered combined with chloroform, one part of chloroform, two parts of ether or two parts of chloroform and three parts ether or combined with alcohol as in the well known "A.C.E." mixture, one, two and three parts respectively of alcohol, chloroform and ether. Billroth's mixture of one part of alcohol, three of chloroform and one of ether is said to be followed by very little vomiting. These are administered in the same method as chloroform alone, the induction being very gradual and no concentration being made. If given on an open mask, I think they are preferable to chloroform alone, so long as it is remembered that chloroform should be regarded as the dominant anæsthetic.

In passing I should like to state that I think it is an unwise procedure during ether administration, especially towards the end of an operation, to use chloroform freely on the mask. A few drops given for a special purpose during full surgical anæsthesia are quite permissible, but to pour on chloroform freely when the patient shows signs of coming out, is I think a procedure not unaccompanied by danger and in almost every instance the required anæsthesia can be obtained by ether alone.

## ANÆSTHESIA AND THE NEWER ANÆSTHETICS: THE INTRATRACHEAL ADMINISTRATION OF ETHER.[1]

By F. W. Green, M.B., B.S. (Melb.),
*Honorary Anæsthetist, The Melbourne Hospital, Melbourne.*

During the past five years it has been my custom to make use of the intratracheal method of administration of ether as a routine in many cases.

This method is of the greatest value in all operations which involve the chance of blood, mucus *et cetera* entering the air passages, as the return current of air makes such entry impossible. It enables the anæsthetist to regulate the depth of narcosis and leaves the field of operation at the disposal of the surgeon.

In intrathoracic operations it provides a positive lung pressure and thus renders the surgeon free from anxiety due to the effect on the lungs of the opening of the chest wall. In operations such as laminectomy and nephrectomy, the postural diffi-

[1] Read at a meeting of the Victorian Branch of the British Medical Association on March 2, 1927.

culties are eliminated. This method also eliminates any local respiratory obstruction from spasm or congestion of the tongue, pharynx or upper air passages. In my experience the after vomiting is much diminished and there is a lessened liability to pulmonary complications, as compared with cases in which ether has been administered by other methods. In my last six hundred cases pulmonary complications occurred in only 0·5%.

#### Technique.

Although there are various forms of apparatus, each possessing merits of its own, they all agree in general principle. There is an ether reservoir which is kept warmed, an instrument for producing an air current and a safety valve. The small machine I have here has been used in the greater number of my cases. It has the advantage of being light and easy to carry and its mechanism is very simple.

The patient to be anæsthetized is prepared in the ordinary way and 0·43 milligramme (one one hundred and fiftieth of a grain) of atropine is given one hour before the operation. It is my practice to give also 0·6 gramme (ten grains) of chloretone on the night before operation and another 0·6 gramme one hour before. I am quite sure that by this means the after vomiting is much lessened.

The patient is now fully anæsthetized by ordinary methods and when completely relaxed is ready for the passage of the catheter. This does at first present some difficulty, but this is largely overcome as confidence is acquired with practice.

The size of the catheter is important. If too large, it may cause subcutaneous emphysema by obstructing the outlet. If too small, it is difficult to maintain an adequate anæsthesia. The average for adults is size 12, for children size 8. The patient's head should be well extended and held in position by a nurse and the mouth opened with a gag. It is advisable to place a folded towel over the upper teeth to prevent injury.

The catheter is introduced by means of Jackson's direct laryngoscope. This is passed over the dorsum of the tongue until the epiglottis comes into view and can be kept well forward by the beak of the introducer. This exposes the opening of the glottis. When the opening between the cords is clearly recognized, the catheter is run in through the laryngoscope, made to enter the glottis and rapidly passed down the trachea. It is essential that the anæsthetist should see the catheter pass through the larynx, as this is the only real proof that the catheter is in the trachea and not in the œsophagus.

Spasmodic coughing and holding the breath often follow the entry of the catheter, but these symptoms soon subside and are replaced by regular breathing. When the anæsthetist is certain of the correct position of the catheter, this is joined up to the efferent tubing of the apparatus. Another short attack of coughing often marks the beginnning of insufflation.

Quiet respiratory movements should go on during the anæsthesia and the patient's face should be rosy and pink. If cyanosis appears it shows that the catheter is too large or has not been passed far enough into the trachea.

During the insufflation the expired air, mucus and so on pass up and out beside and around the catheter, so that no aspiration of blood or other fluid can ever take place owing to the strong updraught.

True apnœa should never occur, although, when the catheter is first introduced some holding of the breath takes place. The respiratory movements, although slight, should be quite definite. A light degree of narcosis can be kept up throughout, for owing to the regularity of the supply of the anæsthetic reflex phenomena do not occur even during a light anæsthesia.

When the operation is completed the ether supply should be shut off and the lungs thoroughly washed out with air to rid them of any ether vapour.

#### Difficulties and Dangers.

Difficulties and dangers are due in the main to some error in technique. The laryngoscope or catheter may injure the pharynx or larynx. When no efficient safety valve is provided or too large a catheter is used, the pressure in the lung may cause over-distension and interstitial emphysema. Liquid ether can be blown into the lung if the reservoir be overfilled and one of the tubes is under the surface of the ether.

The use of warmed ether is essential, since the natural warming arrangement—the passage of the anæsthetic through the mouth and nose—is out of action. When warmed vapour is used, even bronchitic patients are not hampered by ether given by this method.

## ANÆSTHESIA AND THE NEWER ANÆSTHETICS: NITROUS OXIDE AND OXYGEN.[1]

By W. S. Newton, M.D., Ch.B. (Melb.),
*Honorary Physician Attending Out-Patients, The Alfred Hospital, Melbourne.*

Nitrous oxide was first used in 1848 and with oxygen in 1868, but it was until the later stages of the Great War that the extraordinary value of this anæsthetic was generally recognized.

Despite this convincing demonstration of its value very little use was made of this anæsthetic in Melbourne in civil surgery for some years after the Armistice. Other countries, chiefly America, profited more by war experience and were continually enlarging the scope of gas anæsthesia. Not only were nitrous oxide and oxygen frequently employed, but also the efficiency of other gas anæsthetics, notably ethylene, was investigated.

Three years ago I obtained a machine for the administration of nitrous oxide and of ethylene. Dr. Lillies is to deal with the latter anæsthetic and I shall confine myself to the former. During this period I have administered nitrous oxide and oxygen on one hundred and thirty-six occasions with-

[1] Read at a meeting of the Victorian Branch of the British Medical Association on March 2, 1927.

out a fatality, though in the majority of cases there has been serious risk.

### Method of Administration.

I employ the safety gas and oxygen machine which has two reducing valves between the cylinders and the tank and two other valves for controlling the percentage flow of the gases. This machine is also fitted with an ether attachment, so that any desired percentage of the gas can be bubbled through ether if necessary. More modern forms of this machine are also equipped with an attachment for a cylinder of carbon dioxide. This is a complex machine which requires constant supervision and it will not give satisfactory results if employed by anæsthetists who are unfamiliar with its mechanism. In public hospitals its use should be confined to as few anæsthetists as possible, one of whom should be responsible for its maintenance. In public hospitals two to three thousand gallon cylinders connected to the machine by pressure tubing should be installed, otherwise constant troubles will be occasioned by the need of changing small cylinders during operations.

A preliminary injection of morphine 0·01 gramme (one-sixth of a grain) with scopolamine 0·3 milligramme (one two hundredth of a grain) two hours before operation and repeated one hour before is a great aid to successful anæsthesia. This dose must be varied according to the age and weight of the patient. It is the average dose for a normal adult.

The induction may be carried out with nitrous oxide alone or combined with oxygen. I always use the latter method which, though it lengthens the induction period, obviates the feeling of suffocation produced by nitrous oxide.

The depth of anæsthesia is controlled by mixture regulation. I begin with from ten to fifteen parts of oxygen to eighty-five to ninety parts of nitrous oxide and gradually decrease the former and increase the latter until the ratio required for the production of satisfactory anæsthesia in the particular case is determined.

The signs of excess of oxygen in the mixture are superficial and slow respirations, muscle movements or rigidity and phonation. Some patients owing to this error may move and talk during the operation and yet be insensitive to pain and it is not uncommon to find a patient who can repeat remarks overhead during the operation, but who has felt no pain. In normal anæsthesia with a correct mixture, the respirations are full, regular and slightly increased in rate. Phonation does not occur. The muscles are immobile, but retain their normal tone. The eyes are frequently open and the cornea usually insensitive.

If, despite a correct mixture the muscular relaxation is insufficient, this may be increased by one of two methods, namely secondary saturation with nitrous oxide or the addition of ether to the mixture. Secondary saturation is cutting down the oxygen supply to a degree which produces an approach to the fourth stage of anæsthesia. This is said to be a dangerous procedure but Moots[1] and other American observers state that recovery from deep anæsthesia so produced is very rapid if oxygen under slight pressure is administered.

Care is necessary if ether be added to the mixture as comparatively small amounts will produce deep anæsthesia. During ether administration the expiratory valve should be kept open to prevent too great a concentration of ether in the bag. In operations in which nitrous oxide and oxygen are indicated as an anæsthetic, it is rarely necessary to employ ether. Recovery is rapid, complete consciousness being usually reestablished from two to five minutes even after prolonged administration.

It is essential that the mask should fit tightly on the patient's face. If air leaks in under the mask, it is impossible to produce anæsthesia. In the only case in which I failed to anæsthetize the patient, I could not get a mask to fit the face properly.

Rapid induction of anæsthesia and quick recovery are aided by deep respirations which can be caused by increasing the amount of carbon dioxide in the mixture. This can either be produced by the introduction of a percentage of carbon dioxide gas from a cylinder or by keeping the expiratory valve on the mask closed, thus causing rebreathing from the bag.

### Advantages of Gas Anæsthesia.

Gas anæsthesia is by far the safest in skilled hands, though it is admittedly difficult to administer. It produces less effect on any of the bodily functions than chloroform, ether or ethyl chloride. With the latter agents an immediate hyperglycæmia occurs, supposedly by their depressant action on liver and pancreatic function. This disturbance of carbohydrate metabolism may result in a delayed form of acidosis which occurs from twenty-four to forty-eight hours after administration and may be the cause of much subsequent morbidity or even mortality. Gas produces only a mild and transient disturbance of carbohydrate metabolism and does not show even after two hours the gradual depression of the circulatory system which follows the prolonged administration of other anæsthetics. This explains the great value of gas anæsthesia when shock is present.

The advantages of gas anæsthesia are well set out in the following summary by McMechan of the United States of America.[2]

1. The actual duration of anæsthesia is very appreciably shortened in comparison with other methods.

2. Surgical relaxation may be more quickly secured (by secondary saturation or synergists) when desired and as readily dissipated when no longer needed.

3. Recovery of consciousness and rational control are almost immediate, even after prolonged administration.

4. Bodily functions are scarcely interfered with and pathological conditions are not aggravated.

5. By-effects of anæsthetic "hang-over" (headache, nausea, vomiting) are avoided or minimized.

6. Thirst may be quenched and nourishment taken very shortly after operation.

7. Anæsthesia may be repeated several times at brief intervals or oftener at longer intervals.

8. The danger signs of overdose, oxygen want or carbon dioxide depletion are conspicuous enough to give timely warning and the leeway for resuscitation in emergencies is fairly wide. The heart is inclined to "carry on" after breathing becomes embarrassed or stopped and the respiratory centre remains so responsive that often a moment or two of diffusion elimination by withdrawing the anæsthetic or a few breaths of oxygen under pressure or brief artificial respiration suffice to restore vital functions.

The disadvantage of nitrous oxide and oxygen anæsthesia is the absence of complete muscle relaxation produced by chloroform and ether, a disadvantage which is not shared by ethylene.

It is necessary to point out that gentle handling on the part of the surgeon greatly facilitates gas anæsthesia. Such surgeons who resemble engineering mechanics in their methods, will remain dissatisfied with this anæsthetic. Though gas anæsthesia is also strongly indicated in patients who are suffering from shock and hæmorrhage or are in the advanced stages of any infection, my own experience of this anæsthetic has been mainly confined to patients suffering from exophthalmic goître. There is no doubt that in this class of patient nitrous oxide and oxygen form an ideal anæsthetic. The patient comes out of the anæsthetic at once and the convalescence is quicker and easier than after any other form of anæsthesia.

In this type of case the margin of anæsthesia is decreased owing to the fact that the raised basal metabolic rate necessitates an increased oxygen supply. An adequate preoperative treatment therefore lessens the anxieties of the anæsthetist no less than those of the surgeon.

Other types of cases in which nitrous oxide and oxygen anæsthesia has been very effective in my hands, are rib resections for empyemata. Here the mental strain associated with local anæsthesia is avoided and the giving of nitrous oxide and oxygen is not followed by those harmful effects which may accompany ordinary anæsthetics in these cases.

I have also given gas successfully for amputations following diabetic and senile gangrene, for radical amputation of the breast with advanced tuberculosis, for several cases of late intestinal obstruction, for a case of severe secondary hæmorrhage and for many minor surgical conditions.

The cost of nitrous oxide and oxygen anæsthesia is about fifteen shillings per hour. It is to be hoped that this anæsthetic will rapidly become more popular, for it may then be available at a decreased price. Surely this anæsthetic with its quick recovery and safety factor should be the anæsthetic chosen in the casualty rooms and wards of our general hospitals.


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## ANÆSTHESIA AND THE NEWER ANÆSTHETICS: ETHYLENE AND OXYGEN.[1]


By G. Leonard Lillies, M.D. (Melb.),
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Ethylene was first presented to the medical profession in 1923 by Luckhardt and Carter(1) and first employed by Dean Lewis, of Baltimore, and Easson Brown, of Toronto. Ethylene and oxygen were first given in Australia by Dr. W. S. Newton and myself in 1924 to a female, *ætatis* twenty-seven years, suffering from appendicitis. The depth of anæsthesia varied materially. The colour of the patient was ashy grey most of the time and there was profuse sweating—these troubles as later experience has shown were due to an insufficient supply of oxygen. The patient suffered no ill-effects from the anæsthesia and made a normal recovery without vomiting. I had one other unpleasant experience during the early days with this anæsthesia when I inadvertently gave ethylene for a short period with nitrous oxide instead of oxygen; again the patient recovered normally.

### Method of Administration.

Ethylene can be given by means of any efficient gas machine, the one we use being the "Safety Gas and Oxygen Machine." I usually start with eighty parts of ethylene to ten parts of oxygen, increasing the oxygen and diminishing the ethylene according to the colour of the patient and the depth of the anæsthesia required. In the more robust patients it is frequently necessary to give a small amount of ether with the gas during the early stages of abdominal section and occasionally for closure of the wound, but only a very small amount is necessary.

Though I have a very strong aversion to giving morphine prior to ether anæsthesia, I feel that a preliminary injection of morphine 0·01 gramme (one-sixth of a grain) aids ethylene anæsthesia, though I have not yet arrived at a definte conclusion on this point. Induction is always quiet. I have never yet seen any person struggle, even the most robust. The odour of the gas which to the onlooker is unpleasant at first, is not noticed by the patient, consciousness is lost in five or six inhalations, but the period of complete saturation is not reached for from ten to fifteen minutes and it is inadvisable to permit the surgeon to commence the operation until this point is reached. During the earlier stages of anæsthesia the eyes are frequently open and the reflexes are active although the abdominal muscles are relaxed. Later all reflexes are lost and the pupillary changes follow those observed in ether anæsthesia.

[1] Read at a meeting of the Victorian Branch of the British Medical Association on March 2, 1927.

### The Period of Recovery.

Recovery is very rapid, the patient always being able to answer questions before leaving the operation table. It is thus advisable to administer ethylene until the last stitch is inserted. Post-anæsthetic vomiting is rare. Though the patient may vomit a slight amount of clear mucus before leaving the theatre, both ward sisters and hospital residents tell me that the general recovery of all patients who have had ethylene, is very much better than those who have had ether. Two recent cases illustrate this point:

CASE I.—A female, *ætatis* sixty-five years, who had a partial gastrectomy and cholecystectomy was under the anæsthesia for two and a quarter hours. I stopped administration just before the last stitch was inserted. Before the dressings had been applied she was fully conscious and said she felt splendid, but wanted to know when the operation was going to begin. I saw her half an hour later in the ward, she had no vomiting and did not look as if an operation had been done.

CASE II.—A male, *ætatis* seventy-one years, had a partial gastrectomy and removal of part of the transverse colon for malignant growth. The duration of anæsthesia was two and a half hours. He also was fully conscious before leaving the operating theatre and gave no post-operative anxiety for a week. Death took place three weeks after operation from causes unconnected with the anæsthesia.

### Indications and Contraindications.

I have now given ethylene on one hundred occasions and the more I use the gas, the more I am convinced of its value. In my opinion the only contraindication to the use of ethylene is its inflammability. It is a gas having the formula $C_2H_4$, its inflammability being equal to ether vapour or coal gas. It is unwise to employ ethylene when a cautery is to be used. I have used it on two occasions, when the diathermy knife was in action, without disastrous results, once during a gastrectomy and once for diathermy of hæmorrhoids, but I did not feel happy. My only other experience of its explosive powers was an occasion when the rubber bag which contained the ethylene and oxygen mixture burst, due probably to the generating of static electricity in the rubber tubing. This is by far the commonest cause of all reported ethylene explosions and is never, as far as I can make out from literature, very serious as the explosion does not extend beyond the bag and never explodes the cylinders. The risk of explosions from this cause may be overcome by connecting the machine by a wire to earth and by keeping the atmosphere of the theatre moist which is not a difficult matter.

I have never used this anæsthetic in intraoral surgery and imagine that there would be some difficulty in keeping the patient anæsthetized, but Gwathmey, Lundy and others report good results in tonsillectomies and dental operations by using a mask covering the nose only after the patient has been anæsthetized in the ordinary way, then pure ethylene is used, the patient getting a sufficient supply of oxygen through the mouth.

I must confess that at times during my earlier experience with ethylene, I had some disappointing results. Once I failed entirely to anæsthetize the patient and on six other occasions I had to change over from gas to ether given by the open method, because the patient was not sufficiently relaxed to enable the surgeon to proceed. On three occasions the failure was due to a block in the machine. In the other cases, the explanation was a faulty technique. I wish to emphasize two points: (i) That an anæsthetist who wishes to become proficient in the use of ethylene, must not be downhearted at many failures in his early cases, but must persevere. (ii) The necessity of complete cooperation between the surgeon and his anæsthetist. I feel sure the majority of surgeons using ethylene for the first few operations, may be inclined to condemn this anæsthetic, but I wish to make a very strong plea, that they should, like the anæsthetist, persevere and become *au fait* with its extraordinary value.

Lundy holds that a patient who would strain and hold his breath with one surgeon, might be quite relaxed in the hands of another. Undoubtedly patients anæsthetized with ethylene need more gentle handling than is the case with ether. My series of cases include most operations that are met with in a general surgical and gynæcological practice, but by far the greater number have been performed for upper abdominal conditions. Thus I have given this anæsthetic for gastrectomy on seven occasions, for gastro-enterostomy on six occasions, for cholecystectomy on twenty-three and for cholecystostomy once. More than half of these patients received ethylene and oxygen without a trace of ether, the others had varying small amounts of ether and two were given intramuscular injections of 2% "Novocain." Peculiarly enough my best results have been in upper abdominal surgery, though Lundy[2] states that it frequently fails in gall bladder surgery even when ether is administered as well. During one cholecystectomy, when I was giving ether, the patient's condition became so bad just before the gall bladder was completely freed that I changed over to ethylene and oxygen; the patient improved and the operation was completed satisfactorily. The more frail and feeble the patient, the better the anæsthetic result, is a true statement regarding this anæsthetic and I have no hesitation in stating that the surgical risks of this type of patient have been enormously decreased by ethylene.

Ethylene is of great value in genito-urinary conditions, as many of these patients are elderly, with arteriosclerotic, cardiac, pulmonary or renal changes. Further, in cystoscopy when a general anæsthetic is necessary, ethylene has the advantage over other forms of anæsthesia in that it gives sufficient relaxation without diminishing renal secretion.

I have no personal experience of the gas in obstetrical work, but from the rapidity with which analgesia is produced, I believe it will be extensively used, when it is more widely known.

Kreisleman and Kane[3] report eighty-five obstetrical patients anæsthetized with ethylene. Ethylene is given in a concentration of 100% at the onset of each uterine contraction, the patient

being told to take three or four breaths, hold the last breath and "bear down," the anæsthetic being cut off between each pain. This gives sufficient analgesia until the presenting part approaches the perineum when anæsthesia is deepened. It is stated that the duration of the second stage appeared to be shortened. No harmful effects on the child were noted. On the contrary, the promptness with which these babies breathed and cried, was noticeable, as against the sluggishness after ether or chloroform anæsthesia. Owing to the non-toxicity of ethylene, it seems to me to be the most suitable anæsthetic when surgical interference is contemplated in the toxæmias of pregnancy.

Though I have stated that ethylene can be used in nearly every type of surgical condition, this is not desirable in hospital practice for two reasons. The first is the necessity for the instruction of students in the use of ether. The second is the cost of the gas which amounts to about £1 per hour. When the Alfred Hospital first obtained a gas and oxygen machine in 1925, I confined the use of ethylene to those patients in whom anæsthesia was obviously a risky procedure. Now I am giving it in addition to all elderly people, all patients suffering from gall bladder and stomach conditions and those patients who do not fall within Moots's rule for evaluating cardiac reserve. Moots's rule is: The pressure ratio, a fraction having the pulse pressure as numerator and diastolic pressure as denominator, may be normal between 40% and 60%. If the ratio is either high or low, there is reason to apprehend danger. Gilbert Brown[4] states that Miller investigated this rule in one thousand patients and found a death rate of 3·23% among those with operable and of 23·07% among those with inoperable conditions. I have not been using Moots's rule long enough to be able to express an opinion on its value, but the only two patients who have caused me any anxiety since I have been using it, have both been in the danger zone.

I have given ethylene with good results to several patients in my series to whom I would have hesitated to administer ether. The following is an example.

Case III.—A large, flabby male, *ætatis* seventy-one years, suffering from diabetes, aortic regurgitation with heart failure and infective cholangitis with stones in the common bile duct, looked moribund before operation. He took ethylene perfectly and never gave me a moment's anxiety.

Another patient whom I feel would have caused anxiety with any other anæsthetic was the following:

Case IV.—A female, *ætatis* fifty-five years, who had a *fistula-in-ano* and who suffered from advanced pulmonary tuberculosis, had difficult breathing and her colour was a bluish grey when she came to the table. She also gave no trouble.

Alarm may be caused during ethylene anæsthesia by a temporary respiratory and circulatory failure in bad cases.

Case V.—A female, *ætatis* thirty-five years, suffering from intestinal obstruction, was in a very bad state. She was anæsthetized in the usual way with ethylene and oxygen and her abdomen was opened. Before the abdomen could be properly explored her respiration ceased and then her heart ceased to beat. This was confirmed by the surgeon, who massaged the heart through the diaphragm without result. As this patient seemed to be dead, it was decided to close the abdomen with through and through sutures. Two stitches were inserted, when the patient gasped and the pulse became palpable at the wrist, the breathing became regular and her colour which had been an ashy grey, returned to what it was at the beginning of the operation. She had apparently been dead for about three minutes. As she had improved so much, it was decided to continue exploring for the obstruction, which was found and relieved. The patient gave me no further anxiety during the operation, but unfortunately died some hours later.

Case VI.—A returned soldier, *ætatis* thirty years, suffering from pulmonary tuberculosis, had a loose body in his knee joint. Just as the joint cavity was opened, his colour became bad, respiration ceased and the pulse disappeared at the wrist. This condition in this case lasted only about thirty seconds, the respiration and pulse returning in the same way as in Case V.

The probable explanation of the respiratory failure in these cases is that an insufficient supply of oxygen had been given. When the patient's condition led to the removal of the mask and artificial respiration, this oxygen lack was remedied.

That ethylene is an extremely safe anæsthetic, has been recently confirmed by two French investigators, Papin and Ambard.[5]

Donald Guthrie[6] states that Gwathmey has shown that the margin of safety afforded by ethylene is greater than that of nitrous oxide and oxygen, nitrous oxide, oxygen *plus* ether and ether alone.

### Deaths During Ethylene Anæsthesia.

Three deaths in my series of cases have occurred during the administration of ethylene. Please note that I say "during the administration" and not due to ethylene, because, like many anæsthetists, I prefer to look to other causes o fdeath than myself or my anæsthetic.

Case VII.—A young female, *ætatis* twenty years, who was "septic" following an abortion, had been lingering on, as such patients do, for three weeks, draining pus from various abscesses, which had been opened. She was brought to the operating table literally moribund, to have a subphrenic abscess drained. She took only three breaths through the anæsthetic mask and died. I am certain the result would have been the same, if there had been no mask there.

Case VIII.—This death might possibly be said to be due to ethylene. An elderly male, *ætatis* seventy years, had previously had a colostomy done as a preliminary to the removal of a carcinoma of the sigmoid. I saw the patient in his bed before the operation and thought from his appearance that he was dead. In consultation with the surgeon it was decided to administer ethylene and oxygen. The sigmoid was removed and the abdominal wound was nearly closed when the patient collapsed and died.

Case IX.—A male, *ætatis* seventy years, had an irremovable and obstructed carcinoma of the pylorus. A gastro-enterostomy was done and while the skin incision was being sewn up, the patient became cyanosed, in spite of the fact that pure oxygen was being administered. He commenced to struggle and fight for breath, but never regained consciousness, nor did his colour improve. He died five hours after operation and before his death we were able to demonstrate a right sided hemiplegia.

I believe the sequence of events to have been regurgitation from the stomach with inhalation of gastric contents causing asphyxia and cerebral hæmorrhage. I do not feel that ethylene was the cause of death in this case, because all observers are

agreed that there is seldom any alteration in the blood pressure and that when it does alter, there is a slight fall rather than a rise.

### Conclusion.

In conclusion, I must thank you for the patience with which you have listened to me. If I have persuaded any of you to try or to continue the use of what I believe to be the anæsthetic of the future, I shall feel amply rewarded.

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## DR. WILLIAM PALMER, THE POISONER.

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De Quincey in his "Murder Considered as one of the Fine Arts" has exalted certain homicides to a high level. Some crimes by the very courage or intelligence implied in their execution command a certain regard if not respect, but the subject of these notes displayed no laudable attributes. By the promiscuity of his poisonings rather than their subtlety must Palmer take pride of place as Prince of Poisoners and it is a matter of melancholy reflection that he was a medical man. Certainly the nineteenth century could produce no more degenerate scoundrel and Mr. Justice Stephens recorded: "No more horrible villain than Palmer ever stood in the dock."

He placed on the map of England a small town in Staffordshire where Rugely is still known as the place of Palmer's poisonings. After Palmer's execution a deputation waited on the Home Secretary seeking an official alteration of the name of the town, such was its notoriety. "Very well, gentlemen," replied the minister and then naïvely added, "why not do me the honour of calling the town after me?" His name was Palmerston. The matter was allowed to drop. Here it was that Palmer practised the gentle art of medicine until he committed his crowning act of folly and murdered John Cook.

Tennyson Jesse in "Murder and its Motives" places gain or elimination at the head of the list and although these may explain the fifteen or sixteen known crimes of Palmer, it is impossible in the light of modern neurology to consider that he was mentally equipped for social adjustment.

Palmer's grandmother lived and ended her drunken life in a Derby slum. Her daughter, Sarah Bentley, in her teens shared her favours with a humble woodcutter of no education and a man named Hodson, the agent of the Marquis of Angelsea. The woodsman prevailed and rushed her off to a market fair where Sarah became Mrs. Palmer. A pound a week did not go very far even in 1820 so Hodson's attentions were still more than welcome. With a complacent agent Palmer grossly falsified his timber accounts with the Marquis and, having lost his honour, gained riches. The Palmers became carriage folk and in a drab street their house gleamed, as viewed from the pavement, with righteousness and prosperity.

Palmer died suddenly in 1836, leaving behind him some £80,000.

From such dissolute stock came William Palmer, the second of seven children, five sons and two daughters. William was ever the apple of his mother's eye and after his execution she boasted of her "saintly Bill, the best of my whole set." He was a hypocrite from boyhood. Eschewing football, he would rather wander by himself in the fields and secretly dismember insects. Beyond doubt he was always generous and even if this trait was facilitated by other people's money, it never left him.

On leaving school at the age of seventeen, Palmer became associated with a chemist in Liverpool, but irregularities in the cashbox were frequent and prosecution was prevented only when Mrs. Palmer paid a considerable sum. Palmer was bent on acquiring a knowledge of drugs and passed on to an apprenticeship under Dr. Tylecote, of Haywood. Here he met his future wife, Annie Brooks Thornton, and in spite of his notorious association with unfortunate women, he became engaged to her. Throughout his short life Palmer appears to have exercised a peculiar and strong fascination for his various victims. During his five years at Haywood he was known to be responsible for four illegitimate children, three of whom died suddenly soon after birth.

Dr. Tylecote objected strongly to these liaisons and absolutely refused to tolerate thieving, so Palmer had once more to move on, but not before his name had been associated with the sudden death after a drinking bout of a man named Abbey, whose wife had found favour in the eyes of Palmer. This at the age of nineteen. The Staffordshire Infirmary enrolled him as a pupil, but for some reason he was forbidden access to the dispensary. Such restrictions were odious to him and after a few months he left and entered as a student at "Barts." Here his reputation for generosity became enhanced. He was a good fellow, stinting himself in nothing but the payment of his debts. Palmer never drank and was always clean in his language, but in his relations with women he was most intemperate. However, in 1846 he obtained a pass degree and took out his L.R.C.P.

A year later he married gentle Annie Thornton who appears to have been a charming but colourless girl, the illegitimate daughter of a retired army colonel who committed suicide, leaving her 40,000 rupees. They settled in Rugely and for a year his outward respectability lived down his earlier reputation and he collected a fair practice. Shortly after his marriage a Rugely woman brought to the surgery an illegitimate child of his for professional attention. He prescribed, but the patient died within eight hours of the visit. The deaths of his legitimate children were equally unfortunate. Three of the four dying in convulsions soon after birth. The eldest, Willie, survived alone.

Despite the rows of books and anatomical charts that hung around his surgery walls, Palmer was a lazy man. His practice began to suffer. His family was small and even if it suffered an enlargement, it rapidly receded. Had he been contented to leave racing alone, his income amply covered his needs, but, as he jocularly remarked after his trial, "It was the riding that did it." He kept a large racing stable at the edge of Cannock Chase and in 1853 owned six racehorses, but despite an occasional *coup* the fickle dame frowned heavily upon him. If his horse, Nettle, had won the "Oaks" he would have won £10,000, but she bolted, threw the jockey and landed Palmer more heavily into debt.

He turned to his mother-in-law, an eccentric old lady, who after a deal of persuading sent him a cheque for £20. Ostensibly grateful Palmer invited her to live with them. Reluctantly she went, but on her doorstep turned to a neighbour and prophetically remarked: "I know I will be dead in a fortnight." She overestimated her longevity; within a few days her body proceeded a pompous funeral to the Rugely churchyard and nine houses passed to Palmer. He then turned to forgery and during the next two years he raised £20,000 on bills bearing the forged acceptance of his mother. This brought him into the net of an exorbitant money-lender Pratt. During this time and up to his arrest Palmer never failed to attend regularly to the Sunday services and always made marginal notes in his Bible of the salient points of the sermon. Two weeks before his arrest he soundly cuffed a small boy who had dared to giggle during the service.

A man named Bladen was next for the doctor's hospitality. Palmer owed him about £400. Bladen left home on a Tuesday with about £600 in his pocket. Next day after a drive over to Ashley with Palmer Bladen became ill. This was explained by the doctor as the result of the long drive and too much port. On the Friday a complacent octogenarian colleague, Dr. Bamford, was called in and when Bladen died he was quite willing to fill in the certificate under dictation from Palmer.

Bladen's wife had been hastily summoned, but she arrived too late. She was surprised when neither her husband's betting book nor any of his money could be found, but Palmer generously offered to pay all the funeral expenses. Shocked with a dreadful suspicion, Mrs. Bladen left the house, a genuine fondness for Mrs. Palmer stilled her voice.

Palmer at this time was attending a young man named Bly to whom he owed £800. Following a visit from his doctor Bly suddenly died and when the widow mentioned the debt, Palmer explained that she was mistaken, it was really Bly who owed the money, but in the sad circumstances he would not press the matter. Rugely was a small place and yet nothing was done about the frequent sudden deaths among Palmer's associates. Palmer then took out a substantial policy of £15,000 on his wife's life and in some manner managed to get together the first premium of £450. Mrs. Palmer soon after this developed a mild bronchitis and Palmer was most attentive in his care, all her food was taken up by him, with the result that she developed a violent gastro-enteritis. Doddering old Dr. Bamford was called in and as Annie Palmer was too weak to describe her symptoms, her husband accurately detailed the effects of cholera and when she died the certificate was filled in accordingly. £15,000 easy money and Palmer looked round for another suitable life insurance. He decided on his brother who was rapidly drinking himself to death. A policy for £85,000 was suggested and, although the insurance company declined this, a policy for £15,000 was accepted. Palmer supplied his brother with £100 for liquor and hired a servant, known as a bottle holder, to see that he did the sum justice. Nineteen gallons of spirits were soon drunk and in a few weeks later Walter was in a dreadful condition, but not dreadful enough for his doctor brother who finished him off with some prussic acid and gave apoplexy as the cause of death.

When the insurance delayed and eventually refused payment, Palmer must have realized the precipice on which he stood. The pressure of Pratt and the other money-lenders became daily more insistent. He owed in interest alone £6,000 a year and unless he could find this, exposure of his forged bills was certain. With a final dunning letter from Pratt in November, 1855, rose the curtain on the last act of this dreadful drama.

John Parsons Cook, a young man of twenty-eight, cricketer, oarsman and general sportsman, had been left a considerable fortune. What he could not spend on clothing, flash jewellery or liquor, he lost on the turf. By 1855 he owned little beyond a doubtful reputation, the questionable friendship of Palmer and a famous racehorse, "Polestar."

"Polestar" was heavily backed for the Shrewsbury Handicap and won. That night, Cook, Palmer and some of their sporting friends dined at "The Raven." The next day Cook had another win and the dinner was repeated. During the meal a woman called to see Palmer. As she entered the pantry, she saw the doctor dropping a powder into a tumbler which he was holding up to the light and shaking as if watching something to dissolve. Palmer returned to the party with the tumbler. "Polestar" was the toast and Cook, rising with his brandy and soda, drank it straight off. Immediately

he turned to his companion Fisher and groaned: "Good God there is something in it that burns my throat." "What nonsense," said Palmer; "here," to Fisher, "you taste it," which was quite safe, as the glass was empty. Cook hurried from the room and was violently sick. Herring, another member of the party, went to his assistance and Cook, leaning heavily on his arm, gasped: "I believe that damned Palmer has dosed me." Herring remonstrated with him. "You don't know about him" strangely added Cook. And yet this man who knew or suspected Palmer's character, allowed himself to be led off to Rugely and installed in "The Talbot Arms" opposite Palmer's house. It was extraordinary the way this man was able to impose his will on people. The next day, Friday, Cook was a sick man, but with the doctor he went to the races and came back with more than £1,000 in his pockets and an order on Tattersalls for £2,000 to be paid on the following Monday. The success of that day sealed his fate. Cook dined with Palmer, but returned to his hotel very ill. The doctor went with him, put him to bed and suggested calling in Dr. Bamford. While the consultant was examining the patient, Palmer cleaned out Cook's pockets. Dr. Bamford listened quietly to Palmer's account of the bilious attack, ordered some broth and prescribed some pills.

Palmer went home, but in a few minutes the parlour maid at "The Talbot Arms" received from his hands at the door "a bowl of chicken broth for my poor sick friend" intrigued by the fragrance the girl helped herself to a spoonful and as a result persistently vomited all through the night.

Next day, with Cook's notebook in his pocket, Palmer rushed off to London to convert some of the paper. He forged Cook's signature, but his realization fell far short of the amount needed to stave off his urgent troubles. He returned to Rugely, where Cook who had escaped from Palmer's attentions over the week-end, was somewhat improved. This was unsatisfactory, so Palmer called on a chemist friend, Mr. Newton, and purchased three grains of strychnine. That night Cook awoke the whole hotel with his screams and the maid found him "all twisted up in bed." Palmer was called at Cook's urgent request and as he soothed the sick man's hand, he personally administered two more pills and a draught, all of which were promptly vomited and Cook fell asleep.

Next morning Palmer called on the other Rugely chemist, Roberts, and obtained six grains of strychnine, some laudanum and some prussic acid. As he was leaving the shop Newton entered and was naturally surprised when he was told of Palmer's purchase, twenty-four hours after a similar visit to his shop.

Palmer then called on Dr. Bamford and persuaded him to make up two pills for Cook and in his shaky old handwriting to write on the lid "to be taken at once." Rushing out of the room Palmer took three-quarters of an hour to reach Cook's room, about a hundred yards away. Presenting these pills to the room attendant, he drew attention to the old doctor's handwriting on the box and asked for the pills to be given at once. He received this expected summons in half an hour. Poor Cook was screaming with the agony of his convulsions, his body rigidly arched like a bow, but in a few moments, although the contorted face did not relax, Palmer's last victim was dead. Palmer bundled everyone else from the room and sent for a charlady to prepare the body for burial. This lady happened to be downstairs and as she came into the room she surprised Palmer in the act of a final search of the dead man's clothes. Early next morning Mr. Stevens, the victim's stepfather, arrived in Rugely and conceived an immediate detestation for Palmer who did his best to exclude everyone from the affair. Dr. Bamford presented a certificate with apoplexy as the cause of death, but Mr. Stevens when he found that Cook's cash and his betting book were missing, determined on a *post mortem* examination. Palmer had to get busy. He tried unsuccessfully to obtain from the postmaster an attestation to a forged signature of Cook's on a bill for £4,000. He then called on Mr. Newton, the chemist, and enquired as to the *post mortem* findings in a death from strychnine. At this time, in 1855, very little was known of the properties of this drug and the chemist told him that probably no trace would be found. "That's all right," exclaimed Palmer, snapping his fingers.

Palmer attended the *post mortem* examination, which was carried out by Dr. Devonshire, an inexperienced pathologist, who had done only two examinations previously. When the stomach was exposed and looked apparently healthy, Palmer turned to Dr. Bamford, "Well, they won't hang us yet." He appears to have acted in a most foolish and indiscreet manner from the moment of Cook's death. He deliberately pushed Dr. Devonshire's knife into the stomach and nearly all the contents escaped. Some, however, were preserved and together with the intestines were placed in a sealed jar. Palmer managed to obtain possession of this in some manner and slit the covering. Again he was frustrated in time and the valuable packet was handed over to a postboy to be mailed to Dr. Taylor in London. Palmer tried unsuccessfully to bribe this lad with a ten pound note to upset his jar. That night for the first time, Palmer was seen drunk. In a few days Dr. Taylor's report was sent and Palmer persuaded the postmaster, Mr. Cheshire, to open the letter which concluded, "I was unable to find any trace of strychnine." "I knew they wouldn't, I am as innocent as a baby" and his mercurial spirits rose. The postmaster later received a sentence of two years' imprisonment for his action. To crown his remarkable conduct, Palmer sent to the coroner, Mr. Ward, a generous present of some game and a ten pound note, enclosing a note in which he unwisely let out that he had seen Dr. Taylor's report. "I hope the verdict tomorrow will be a death from natural causes and thus end it." Next day Palmer entered the court full of hope, but in spite of the bribe Mr. Ward brought in a verdict of wilful murder and Palmer was committed to prison. The

case became the talk of England. The body of Mrs. Palmer was exhumed and found to contain antimony; again a verdict of wilful murder was brought against Palmer. His house was searched and a book on toxicology was found with the page on strychnine turned down. Such was the local prejudice that after some delay a special Act of Parliament was passed, *The Palmer Act,* to enable the trial to take place in London.

On May 15, 1856, at the Old Bailey, before Lord Chief Justice Campbell, Baron Alderson and Mr. Justice Cresswell, William Palmer was charged with the wilful murder of John Parsons Cook by means of strychnine. So great was the universal interest, that other High Court Judges attended to listen to the proceedings. Leading for the Crown was one of the most able advocates of the time, Sir Alexander Cockburn, Attorney-General. His performance in the Palmer trial was considered his supreme achievement. Three years later he became Lord Chief Justice. With him were four leading barristers.

Sergeant Shee was at the head of a talented legal team of four appearing for the defence. Cockburn produced a mass of circumstantial evidence that strongly withstood a severe cross-examination, but when he came to Dr. Taylor's evidence it was admitted that no strychnine had been found, but a small trace of antimony was present. He gave it as his opinions that judging from the clinical symptoms Cook's death was due to strychnine poisoning. Then for the next few days ensued a most intricate medical discussion. Dr. Taylor was severely handled by Sergeant Shee. Dr. Herdpath for the defence heatedly accused Dr. Taylor of incompetence and justly brought on his head a rebuke from the Judge, "It is indispensable to the administration of justice that a witness should not be turned into an advocate." No less than twelve medical men were called for the defence to refute the expert evidence of the Crown, but the strong circumstantial evidence was untouched. The whole question was whether the tetanic spasms were the result of poison or disease. The Crown held to the former, the defence held that Cook died as a result of disease. The infective nature of tetanus was not realized at that time and medical evidence was given that the condition might arise from natural causes. Mr. Curling, a reputable member of the Royal College of Surgeons, who had written a book on tetanus, gave evidence that idiopathic tetanus was apparently self generated. Sergeant Shee called evidence that Cook suffered from syphilis and the Crown produced evidence of the absence of syphilis. Palmer's repeated purchases of strychnine, his extraordinary behaviour before and after and during the inquest, his dire financial position, these were not discussed by the defence. The final speech for the Crown was a masterpiece. Even Palmer leaned forward, fascinated and absorbed. The summing up of the Lord Chief Justice was prepared during the fourteen hours of a Sunday. He pointed out that there was no rule of law according to which the poison must be found in the body of the deceased to bring about a conviction and although the address was impartial to the evidence, it appeared to weigh against the prisoner. At the end of the fourteenth day of the trial the jury retired to consider their verdict. At the end of ninety minutes they arrived at a unanimous opinion of Palmer's guilt.

That evening, handcuffed, Palmer passed through Euston Station and in the train his legs were chained to one of the warders. Attempts to appeal were started, but were dropped. It was still the time of public executions and one fine morning in June in Stafford Street, surrounded by a cruel howling mob of over thirty thousand people, hurling vile names, Palmer stood calmly and as the rope was adjusted he firmly said, "I am innocent of poisoning Cook by strychnine."

So died this dreadful man, not in old age, not even in middle life, but at the pathetic age of thirty-one. Within a few days a notice appeared in the front window of Palmer's house:

PHOTOGRAPHIC PORTRAITS.

C. Allen

Respectfully informs the ladies, gentry and inhabitants of Rugely that he can produce a very superior photographic portrait in gilt and other frames from one shilling to one guinea and solicits their patronage at the rear of the premises lately occupied by

WILLIAM PALMER.

## FOOD SUPPLY FOR NATIVES IN THE TERRITORY OF PAPUA.

By W. M. Strong, M.D., B.Ch., D.T.M. & H. (Cantab.),
*Chief Medical Officer, Territory of Papua.*

The leading article in The Medical Journal of Australia for September 4, 1926, has been brought to my notice and it has been suggested that it might be interpreted as indicating that Australia through her dependency the Territory of Papua has been remiss in the matter of attending to the wants of the quarter of a million Papuans which Australia has been responsible for during the last twenty or so years, or in the alternative that the Papuan Administration has been so remiss. I do not for a moment suppose that this was in the mind of the writer of the article in question, but it does seem to me that those not conversant with all the facts might so read the article. More particularly I would like to urge that whilst it is a moot question as to whether or no the population of Papua is slightly increasing or diminishing on the whole, yet the Papuans are in no sense a race which is dying out. Certainly in some districts the population is diminishing and in others increasing. My own personal opinion is that it is mainly a question of food supply, including an adequate supply of the various vitamins. Certain phases in the leading article would almost suggest that until the article by Dr. Cilento (which was the subject of comment) was published, little or no attention had been paid

in Australia or in Papua to the inadequacy of native diets and especially the inadequate diets which signed-on native labourers are allowed.

As a matter of fact at least three of the medical men in the Territory of Papua recognized the importance of paying attention to native ration scales and the importance of vitamins in the matter of beri-beri and scurvy as long ago as 1911. At that time valuable work was being done on the question in Manila and this work was sufficient to convince Doctor Jones, then Government Medical Officer at Samarai, Papua, myself and Doctor Goldsmith, then Chief Medical Officer, Papua, that a suitable diet was the one important factor in preventing beri-beri. The history of a patient in the early stages of beri-beri whom I treated at Cape Nelson in 1910-11 served pretty well to convince me and also Doctor Jones that the views propounded by the Americans in Manila worked out in practice. This case is referred to on page 156 in the Annual Report for the Territory of Papua (for the year ending June 30, 1911) in the following words:

> Only one case of beri-beri came under observation this year, although a serious outbreak occurred at Woodlark. The one case seen here was sent in from Cape Nelson and as treatment along the lines which have been found so wonderfully successful in the Far East (referred to in last year's report) had been instituted before the patient left Cape Nelson, he was almost convalescent when he arrived at Samarai.

Scurvy has for more than a century been recognized as a diet deficiency. Scurvy, especially in the form of so-called "sore mouth," was for long recognized in the Territory. By paying more attention to the feeding of native labourers and prisoners it has been pretty well banished from the Territory.

During the years of the war it became recognized by me that certain forms of eye trouble in Papua leading to general panophthalmitis were dependent on a diet deficiency (the or perhaps one of the fat soluble vitamins of the "A" group being deficient).

The whole question of the feeding of native labourers was gone into with the larger employers of native labour in 1921 and fully reported in the Annual Report of the Territory of Papua for the year 1920-1921 on page 100 *et sequelæ*. I would like to express my appreciation for the way in which the larger employers of labour received the advice I gave them. The following extract is quoted from this Annual Report:

> The rations which natives in employment, or in gaol, ought to receive has always been a matter on which different people have held different opinions and this matter has been given a good deal of attention during the year.
>
> There are several factors which have to be considered in making out a ration scale. The total quantity of food must be sufficient. Some of this food must be in the form of proteid and, if European standards are adopted, a fairly large proportion of proteid must be given (something over 100 grains a day). But recent work has shown that at least three further constituents are necessary for satisfactory health. These three other constituents are called vitamins. If one of these constituents are absent or deficient from the diet over a long period, sickness is likely to ensue. If the first of the vitamins are deficient, beri-beri is likely to occur, while if the second is deficient forms of scurvy are also likely to occur. Deficiency in the third vitamin does not so clearly produce disease; but ill-health and eye troubles are likely to occur if this vitamin is deficient and in the case of immature animals or human beings growth is affected. One can confidently assert that there are other constituents of the food eaten which are necessary; but it is a matter on which exact knowledge has not yet been reached.
>
> Whenever there are a large numer of men living what is usually termed "barrack life," a demand arises for a ration scale. This, I suppose, is inevitable, but I feel almost tempted to say that no ration scale can be entirely satisfactory. It seems clear that there are so many constituents of the food necessary for continued good health that it is pretty well impossible to effect this unless a varied and changing diet is allowed.

I think the larger employers of native labour influenced the smaller. In point of fact there was no further serious trouble with deficiency diseases for a long time. A new company, however, started and was very much inclined at first to ignore this matter; perhaps influenced by the impossibility of always obtaining unanimity in the profession in these rather specialized matters. However, beri-beri, scurvy and eye troubles broke out amongst their labourers. All trouble very promptly ceased when attention was paid again to the food supply.

Following on this trouble and early in 1926 the matter was again under consideration and two pamphlets written by me were published by the Government. I shall be very pleased to send copies to medical men who write to me and ask for the same.

One pamphlet was entitled "The Feeding of Native Labourers in Papua" and was primarily written to influence private employers of labour and Government officers and departments employing free or prison labour. The other pamphlet was entitled "Improving the Food Supplies of Native Villages." It was mainly written with a view to inducing missionaries, Government officers and perhaps others having an influence in native villages to induce the village natives to grow a more suitable variety of foods. Perhaps I should add that it was hoped also that those in authority would continue to remember that from the point of view of the village native his food supply is his first consideration and that if road making, house building and the thousand and one new activities which the advent of Government officials, missionaries and traders tend to impose on the native, lead to inadequate attention being paid to the food supply, then disaster to the population is at hand. I would like to quote here the first paragraph in the introduction to this pamphlet:

> I suppose everyone is aware that a person's health and condition, both physical and mental, depends very largely on what he eats and drinks. Recent medical work of a scientific nature has supplied much new information regarding this matter. It is gradually becoming more and more apparent as an important bearing on the question of native food supplies not only on the plantations but also in the native villages themselves. The influence is not merely an academic one but would appear to affect appreciably not only the death rate but also the birth rate.

The question of these deficiency diseases was also referred to at some length by me at the recent Inter-

national Pacific Health Conference, Melbourne, December, 1926. The matter was summarized substantially in the following words:

Such deficiency diseases are not prominent in the native villages—at all events in a clear and evident form. Beri-beri would seem to occur in sago-eating districts at times. But, apart from this, I believe that natives often have their resistance lowered due to a relative deficiency of vitamins in the diet. When we leave the native village and come to consider the native living in labour camps or in gaols and living on a diet with little or no fresh food, the condition is quite otherwise. Beri-beri was once very rife under such conditions. Very acute cardiac cases and cases of dry beri-beri were once common under such circumstances. The replacement of a portion (one-third) of the rice ration by wheatmeal has led to an enormous improvement in this respect. Ulcers and especially ulcerated conditions of the mouth accompanied by spirochetes and fusiform bacilli have often under such conditions caused much trouble. I regard these ulcerated conditions as being scurvy in the sense that a deficiency in the diet of the anti-scurvy vitamin "C" leads to a susceptibility to such conditions. Such cases (often in epidemic form) certainly clear up satisfactorily only if fresh food is added to the diet, or germinated peas given. We have had epidemics of eye troubles leading to panophthalmitis and total destruction of the eye. Such cases are, I am satisfied, due to a deficiency in the fat soluble vitamin "A." I rather suspect that it is mainly the lack of this vitamin which makes so many native races susceptible to serious tubercular infections. Perhaps it is more than a coincidence that following on an epidemic of eye trouble in one centre of native labour employment, there are now signs of an undue proportion of tubercular cases. Apart from these cases of real disease, I am confident that many natives suffer in the matter of general resistance to disease and are unable to work at their full efficiency. Even in the villages I think this occurs. Modern research is, I think, tending to show that a vitamin (of the "A" fat soluble group) is necessary for reproduction. In Papua there are districts where even our very crude and perhaps often unreliable statistical information would seem to show that the population is definitely diminishing. It would seem that in such districts it is the birth rate which is at fault. The death rate is high, but on the other hand no higher than in districts where the population seems to be definitely increasing. I rather suspect that the lack of a vitamin similar to the fat soluble one has much to do with the matter. With regard to tuberculosis I would like to add that every medical officer when he first arrives in the country seems to take it as almost axiomatic that tuberculosis is going to be the great health problem in the country in regard to natives. I thought so myself twenty-three years ago when I saw there were cases in the villages and that a large proportion of the mission teachers then showed suspicious signs of tubercle. However, in the villages generally the travelling officer's reports indicate no particular increase of this trouble. There is, perhaps, some slight increase in the villages which are in closest contact with Europeans and where there is most tendency to eat over-milled rice and white flour in place of native vegetables. In this matter I would like to quote from "The Practice of Medicine in the Tropics," edited by Byam and Archibald, Vol. II, page 1197:

> The records of the Health Office at Charleston, S.C., extend from the year 1822 to the present time. By reference to the chart compiled from these records (Fig. 443) it is seen that the mortality from consumption for the period 1822 to 1830 was high but nearly equal in the white and in the coloured populations, and that it declined in latter decades but still remained nearly equal. From 1865 on, however, the difference between the two races becomes enormous. On the whole the white death rate continues to decline but that of the negro shoots up to become two or three times as great as that of the whites and there is no longer the marked parallelism of the fluctuations of the two rates.

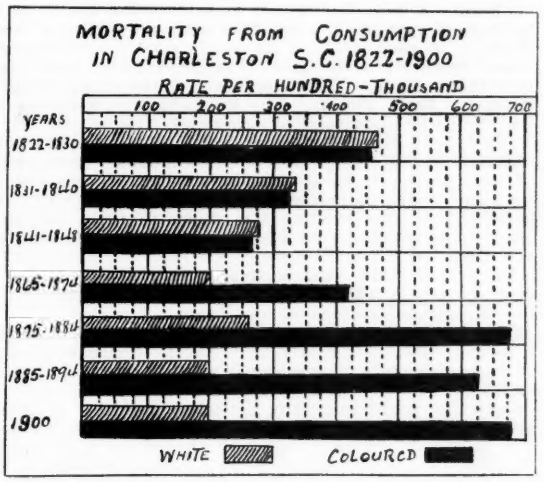


Figure referred to by Dr. Strong from "The Practice of Medicine in the Tropics," by Byam and Archibald.

The tables show the comparative incidence of tuberculosis amongst negroes and whites in America..

In the tables you will see that while the negroes were slaves (in other words while they were a sufficiently valuable a commodity to be carefully housed and fed) they showed no special liability to tubercle. As soon as they were free and had to provide their own food and housing, they showed a marked susceptibility to tubercle. In fact, it appears to me that the tubercle problem is largely one of ordinary feeding and hygiene. I think it is futile to think that we are going to keep a race under modern conditions out of contact with tubercle. We must try and arrange matters that they gradually acquire a partial immunity to tubercle similar to that which Europeans possess. I would, however, do all I can to diminish the probability of what I may call massive infections.

Of course our revenue is not adequate to do anything I would like to in the matter of health and medical work in Papua. We are not a rich country like Australia and have to do the best we can with the resources available. But, still, I suppose lack

## Reports of Cases.

### SOME OPHTHALMIC CASES.

By J. C. Douglas, L.R.C.P. & S. (Edin.), D.O. (Oxon.),
*Honorary Ophthalmic Surgeon, Ballarat Hospital, Victoria.*

The following are medical or surgical cases with an unusual ocular interest, seen recently in the wards of the Ballarat Hospital.

#### Brain Tumour.

Mrs. E. M., *ætatis* fifty-nine years, was admitted on May 1, 1926. Her illness was of six weeks' duration. The symptoms were persistent headache, weakness, increasing weight, mental dulness. No vomiting was present and the eyesight was clear. On May 12 the *fundus oculi* was examined on both sides and presented no abnormality. On May 18 there was a moderate degree of optic neuritis present in both discs; the discs had blurred edges and one small hæmorrhage was present on the right disc and two small hæmorrhages on the left disc.

On June 3, 1926, the clinical notes recorded double optic neuritis of extremely severe type with a swelling of four diopters in each eye. Many small striate hæmorrhages were present in both discs. The pupils were equal and moderately dilated. The appearance of the fundi strongly suggested brain tumour. The headache was more severe and the patient had vomited only twice.

On June 9 Dr. Sloss performed a right and left subtemporal decompression by Cushing's method; large areas of bone being removed. No sign of tumour was found. On June 18 the optic neuritis appeared unchanged in its severity, but the swelling was down to three diopters. Many hæmorrhages were seen, but no white effusions or star figure.

On July 7, 1926, *post mortem* examination revealed a large degenerating hæmorrhagic glioma in the right temporo-sphenoidal lobe of the brain.

This progressive development of an optic neuritis from a normal disc to an extremely severe textbook picture with four diopters of swelling in a little over a fortnight seemed to me extraordinarily rapid for a brain tumour, though this might be seen in secondary syphilis. It might also possibly be seen in acute disease of the sphenoidal and posterior ethmoidal sinuses, though in these cases the condition is usually an acute retrobulbar neuritis, as in a typical case which I saw last year in the Ballarat Hospital. On the other hand the development of papillœdema of the disc may be extraordinarily slow in brain tumour. I have seen a case in which there had been suspicious, vague, indefinite signs of optic neuritis, with some equally vague other signs over a period of fifteen years. Then suddenly the condition lighted up into a flaring optic neuritis with every sign of brain tumour. A tumour of the mid-brain was found *post mortem*.

In another patient whom I examined a year or two ago, there seemed to be a moderate degree of optic neuritis. This patient had been under observation for twelve years and the condition was just commencing to get more definite. I do not know the subsequent history, as he was not my patient.

At times the fundus picture in brain tumour is quite atypical. Some little time ago a young girl was sent to me for a fundus examination. She had a typical albuminuric retinitis, yet repeated testing proved the urine to be normal. Subsequently her condition was diagnosed in a metropolitan hospital as due to a brain tumour. She was operated upon and death ensued. Optic neuritis is more likely and more severe in tumours of the mid-brain, cerebellum and temporo-sphenoidal lobe; while it may be absent in tumours in the pituitary fossa, the white area of the cerebrum and the medulla.

### Multiple Bone Disease Causing Bilateral Proptosis.

Miss M.B., *ætatis* sixty-one, was admitted on November 8, 1926. There was a history of osteomalacia all her life, many fractures occurring between eight and fourteen years of age, then less often in adult life, but some occurred as late as forty-eight, forty-nine and fifty-four years of age. These involved the ribs, both forearms, left thigh, both clavicles and the right humerus twice. Twenty years ago she developed septic bone disease of the lower jaw from a tooth and first the left half and then the right half of the jaw was removed by operation.

She has had a discharging sinus of the left upper jaw of two years' duration. For one year she has had moderate proptosis of both eyes, more pronounced on the left side where the eyeball is displaced somewhat up and out. Diplopia is present. The eyes have always been a little prominent. On November 10, 1926, it was noted that moderate exophthalmos was present, the left eye was displaced up and out, diplopia was present, the optic discs were pale and vessels showed arteriosclerosis. A bony tumour could be palpated inside the left lower orbital margin. On November 11 limitation of movement of left eye to the left was noted, there was a wide palpebral fissure, defective convergence was prominent, the brows did not wrinkle when she looked up, Graefe and von Stellwag's signs were not present. The X ray report indicated that the left antrum was filled up by a bony mass, but the report was otherwise indefinite. The X ray picture did not clearly bring out the bony masses which could be felt in the orbits.

On December 3, 1926, there was œdema of the bulbar conjunctiva and lower lid on the left side; incision and drainage were carried out.

On February 5, 1927, she was discharged relieved. Proptosis was still present, but no diplopia and no displacement of eyeball. Some discharge was still coming from the alveolar region on the left side.

The pathological condition was probably one of caries of the bone with an inflammatory proliferation and thickening in the outer layers of the bone, amounting in places to an actual outgrowth as in the orbits.

It is an unusual cause of proptosis and does not occur among the series of very interesting proptosis cases recently reported in this journal by an Adelaide oculist.

### Purpura Hæmorrhagica with Retinal Hæmorrhages.

Miss S.R., *ætatis* ten years, was admitted on January 5, 1927, with *purpura hæmorrhagica*. In addition to subcuticular spots there were hæmorrhages from various mucous membranes and internal organs.

On January 6, 1927, the *fundus oculi* in each eye was the site of numerous round and irregular hæmorrhages of moderate size in the central area, but not beyond this. Some of the hæmorrhages were horseshoe shaped and others presented a heaped up darker centre, but none manifested the pale centres due to leucocytes, described by Foster Moore as characteristic of leuchæmia and some other blood diseases. Some patches were striated at their edges. Most of them were close to or surrounded a vessel. The left fundus was the site of an elongated subhyaloid hæmorrhage extending from the disc to below the macula. The entire fundus was quite pale. The disc edges were clear. Vision was little affected.

On March 3 according to the clinical notes the fundi were paler than before. Some fresh hæmorrhages had occurred. Many of the patches now had pale centres. The whole fundus had a mottled appearance in its central area. The disc edges were blurred. The vessels seemed half empty and had a beaded appearance in places; they were so pale as to be hardly visible at times. It was difficult to follow them on the disc. The large subhyaloid hæmorrhage, previously noted, was still present, but had broken up. There had been some small subconjunctival hæmorrhages in the previous two months which were quickly absorbed.

The retinal changes in this disease have not been reported in any detail and are barely touched upon even in books on medical ophthalmology. The ocular findings are alone given here, as the whole case will probably be reported in all its aspects by the Medical Superintendent of the Ballarat Hospital.

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## ECTOPIC PREGNANCY IN THREE SISTERS.

By William L. Crowther, D.S.O., M.B., B.S. (Melb.),
*Hobart, Tasmania.*

The three cases that I wish to describe briefly, are of great interest inasmuch as they introduce a factor into the causation of ectopic pregnancy that I do not remember having seen mentioned before. It will be seen that ectopic pregnancy occurred in each of three sisters, the diagnosis in each case being subsequently confirmed at operation. Such a series must give rise to the possibility of a congenital or familial cause being present. Accurate and detailed notes and case-sheets are not available in any of the three instances, but the following brief histories give some of the essential details of the cases.

On April 3, 1926, I was called to see Mrs. H.C., *ætatis* twenty-three, who was complaining of a very severe abdominal pain. She stated that two days previously she had had an attack of very bad pain low down in the left side, which had gradually subsided. She had one child, aged two years. She was then some fourteen days over her time of menstruation and had had some little blood-

stained discharge from the vagina. Vaginal examination revealed nothing except definite tenderness towards the left side. A diagnosis of a ruptured ectopic pregnancy was made and operation advised.

On section free blood was found in the peritoneal cavity. The uterus a little enlarged and a rupture of the left tube found at the junction of its middle and outer thirds. The right tube and ovary did not appear to be in any way abnormal, except that the tube was perhaps a little shorter and of a slightly greater diameter than is usual.

I did no more than remove the left tube, clean out the effused blood and close the abdomen.

Her convalescence was an anxious one, as she had much cyanosis, dyspnœa and cough with a copious watery secretion from the lungs for several days. She had two small pulmonary infarcts about the ninth and eleventh days. On taking her history it was ascertained that two of her sisters had been operated upon for the same condition. The first, Mrs. H., was thirty-five years of age. One child, eleven years of age, had died a year previously of congenital heart disease.

On October 23 I saw her in the absence of her own medical attendant, Dr. W. I. Clark. She was then lying on the floor in intense pain and had vomited copiously. The pain was referred to the right side and very low down. She had menstruated for four or five days, finishing on October 14.

She had returned to her home from Sydney just six weeks previously. There was no history of any vaginal hæmorrhage. I considered the onset to be too acute for appendicitis and made the diagnosis of ruptured extra-uterine gestation. Dr. Clark operated upon her two days later and I was present as anæsthetist. Again free blood was found on opening the peritoneum and a rupture of the tube at its extremity, the pregnancy being tubo-ovarian in type. The extravasation of blood was surprisingly little in view of the great severity of the initial signs and symptoms. Mrs. H. made an uninterrupted recovery and has since been delivered normally of a child.

The third sister, Mrs. H.H., *ætatis* forty-one, was taken ill at 11.30 p.m. on March 28, 1924, after having just returned from the cinema. She describes the pain as having been all over the stomach, very severe and of a bearing down nature. She gave a history of having had one miscarriage some twenty years previously. For two or three months before her attack she had not been regular in her menses and suspected she might possibly be pregnant. Her condition was so bad that immediate removal to hospital for operation was effected. At 6.30 a.m. an abdominal section was undertaken. Her husband was told that her abdomen was full of blood and that her condition at that time was very grave. The hospital records state that there was a ruptured ectopic gestation of the right side. A double salpingectomy and left oophorectomy were done, after which she made a good recovery.

### Commentary.

I am unaware as to whether such a series of cases in the one family has been reported before. In my own limited experience I have never heard nor read of there being any family tendency to such a condition. The mother of the three women concerned has had eight children and does not remember having had any abdominal crisis, suggestive of such an accident. All three women are well above the average in general physique and health. No Wassermann test was done, nor did there appear to be any circumstances to render such test desirable. There was no history of leucorrhœa. In neither of the two cases with which I was associated at operation, did there appear to be any sign of oldstanding or recent adhesions of the uterus or its adnexæ.

It seems then as if one may exclude syphilis and gonorrhœa as causal factors in these three cases.

A history of simple inflammation or such subsequent to pregnancy or miscarriage, if it had taken place, was not remembered.

It is to be regretted that specimens for microscopical examination were not taken; as valuable information as to the cilia *et cetera* might have been obtained.

The tubes did not appear to be infantile, nor were they convoluted in any such degree as to suggest infantilism. In the instance of Mrs. H. C. I noted particularly that they were short, straightened and perhaps of an increased diameter.

No diverticulæ nor accessory tubes were seen.

It may be then that some essentially family factor of type and character, unrecognized and unknown, is responsible. Such a condition as a relative acceleration of trophoblastic development would be a possible cause.

Such considerations must be left to those who are better qualified than myself to judge, my reason for presenting these notes being the hope that they may be of use to others of wider experience.

## SEQUEL TO FRACTURE OF THE ELBOW.

By S. Goldberg, M.B., Ch.M. (Sydney),
*Griffith, New South Wales.*

The following complication of a fractured elbow might be worth recording.

Five months ago I was called to see a girl, aged ten years, who had fallen on her elbow. I diagnosed a fracture of the medial epicondyle and under an anæsthetic set the elbow in the position of acute flexion. Passive movement was begun in a fortnight and after the usual treatment of massage *et cetera* she obtained perfect movement at the elbow joint.

Three months later she returned with a typical *main en griffe* and showed the following signs: (i) Anæsthesia to light touch on the medial side of the front of the wrist and palm and the little and half the ring fingers. (ii) Paralysis of the intrinsic muscles of the little finger and wasting of the muscles on the medial aspect of the hand.

I applied diathermy by the plate method, that is one plate over the medial aspect of the elbow and the other on the lateral aspect. A current of eight hundred milliampères for a period of thirty minutes was applied every alternate night.

After four weeks of this treatment there was complete recovery of both the sensory and motor functions of the ulnar nerve.

My conception of the case was that the callus thrown out to unite the medial epicondyle to the shaft of the humerus, caused pressure on the ulnar nerve causing the signs described above. The induced passive hyperæmia of the diathermy current must have removed the excess of callus.

## CARCINOMA OF THE RIGHT LUNG.

By H. T. Illingworth, M.B., Ch.M. (Sydney),
*Honorary Medical Officer, Auburn District Hospital, New South Wales.*

On October 20, 1926, I was consulted by the patient, J.O'S., a builder's labourer, aged thirty-three years, of fair complexion and slight build. He complained of a tight feeling in the right mid-clavicular and left parasternal regions, about the levels of the third to the sixth intercostal spaces. The duration was only three weeks. His previous history was quite normal, except that he had had "influenza" six months previously and this had left him without his seeking medical advice for it.

He stated that he had never felt quite himself since the attack, but had been able to resume his work and had been able to do his work quite well, until the present attack commenced.

His only other symptoms were a slight cough, with a little white frothy sputum not bloodstained, a poor appetite, a feeling of tiredness and weakness on exertion and the loss of weight, which he stated to be about 3·1 kilograms (seven pounds) in the previous three weeks.

On examination he looked a sick man, his cheeks were thin, sallow and pale and he had an anxious expression

with some dyspnœa on any exertion. He had no fever, a pulse rate of ninety while at rest, a furred tongue and some trembling of the hands.

His urine was clear and no tubercle bacilli could be found on examination. Examination of his abdominal and nervous systems revealed nothing abnormal. His heart sounds were clear, but weak, the apex beat was displaced downwards one intercostal space and outwards to just outside the nipple line. His pulse was of poor volume, the blood pressure was not taken. His breath sounds were not considered abnormal in the left side of the chest, but at the right apex and at the right cardio-phrenic angle bronchophony and in places pectoriloquy were found. There were also increased vocal fremitus at the same areas, many medium friction rubs all over the right side of the chest in front and a few moist sounds in scattered places. Posteriorly nothing else was made out. I decided to have the chest examined by X rays for evidence of pulmonary tuberculosis or other lesion. I also had a specimen of the sputum sent to the Board of Health for detection of tubercle bacilli or other organisms.

Three days later with rest and a stimulant form of diet and drug treatment he was not improved.

On October 26, 1926, the Royal Prince Alfred Hospital reported as a result of X ray examination that there was a small, dull area with a well marked cavity at the right cardio-phrenic angle and that this was almost certainly due to bronchiectasis. No signs of tuberculosis were seen. The Board of Health also reported that there was no evidence of tubercle bacilli being found in the sputum. The usual treatment for a bronchiectatic cavity was given, various types of inhalation and other means such as liniments, "Antiphlogistine" *et cetera* without very much benefit.

On November 7, 1926, the patient complained of distress whenever he went to lie down and the sputum had become thicker and bloodstained.

As his symptoms were unrelieved, I decided to send him into hospital, where he could be given hypodermic injections and other treatment more efficiently. He was admitted to the Auburn District Hospital on November 9, 1926. I am indebted to the Resident, Dr. J. J. McIntosh, for subsequent notes till his transfer to the Lidcombe State Hospital. Dr. McIntosh reported that the patient complained of pain in the upper part of the right side of the chest and of paroxysms of coughing, accompanied by discharge of much phlegm. As the illness progressed, he could not take food, because it caused vomiting. On admission, the temperature was 38.3° C. (101° F.) the pulse rate 120 and respirations 36 per minute. He was pale, had an anxious expression, the breathing was very distressed, all accessory muscles being used to expand the chest. There was definite expansion of the left side of the chest, but little if any on the right. Intense respiratory effort interfered with his speech. Any movement caused distress.

On physical examination there was the lack of expansion on the right side noted above, no vocal fremitus was present on the right, except just below the clavicle. Percussion revealed a highly resonant note over the right clavicle down to 3·75 centimetres (one and a half inches) below it. The whole of the remainder of the right side both anteriorly and posteriorly gave a dull wooden sound. On auscultation cavernous breathing was heard just below the clavicle, no breath sounds could be picked up over the rest of the lung, except from about ten centimetres (four inches) downwards from the apex. Vocal resonance was greatly diminished. In the left lung scattered areas of consolidation were apparent. No abnormality of any of the other systems was revealed by examination.

On the following day in consultation with Dr. J. A. Lawson we decided to aspirate the chest, 1·3 litres (two pints, six ounces) of thickly bloodstained fluid being drawn off, with great relief to the patient who had been extremely distressed since admission with fits of coughing and vomiting. The sputum was clear and tenacious, with small, greyish-yellow masses here and there. It was considered inadvisable to take any more fluid from the chest as there appeared to be a hæmorrhage going on into the pleural cavity—in fact a hæmothorax was present.

The patient was accordingly given 0·6 gramme (ten grains) of calcium lactate three times a day and "Hæmostatic serum" five cubic centimetres twice a day. Also morphine sulphate fifteen milligrammes (one quarter of a grain) was given with atropine. The coughing seemed a good deal relieved, but the patient stated that he felt as if his chest was filling again. On November 15, 1926, the patient's condition still seemed very bad.

Chest signs varied from day to day, tympanitic resonance with cavernous breathing giving place to dulness with absence of breath sounds at the right clavicular region.

On November 16, 1926, it was thought advisable to allow a specialist to give an opinion and Dr. A. J. Collins, of Macquarie Street, saw the patient with us in consultation on that day. No assistance to diagnosis had been found from an examination of the aspirated fluid or in the sputum and Dr. Collins gave his opinion that the condition was either pulmonary tuberculosis or malignant disease. He advised further rest and if necessary another aspiration after a few days. *Linctus morphinæ* was added to the same treatment which was continued. The temperature which had fluctuated between 38·9° C. (102° F.) and 37·2° C. (99° F.) since admission, gradually became normal and the pulse rate also dropped. On November 16, 1926, the urine was found to contain pus cells. This cleared up in a few days without leaving any trace.

By November 20, 1926, the patient appeared to be much improved, but a very guarded prognosis was given. However, the patient lost his anxious appearance, began to eat better and the breathing became less laboured. The area below the right clavicle became tympanitic and the sputum almost ceased. We felt inclined to think that the hæmorrhage had ceased and that we were dealing with a tuberculous cavity in which a large clot had formed. This seemed to be borne out by the large area of dulness which could be mapped both anteriorly and posteriorly over the right lung. The improvement continued and we thought it advisable to suggest a transfer to some more convenient place where patients with chronic diseases were accommodated. In consultation Waterfall Sanatorium was suggested, but it was finally decided to place the patient nearer his friends and on December 1, 1926, he was transferred to the Lidcombe State Hospital.

Complete notes of his case were forwarded there and on admission he was carefully examined. The following day he developed some dyspnœa and on that evening the Resident (Dr. Boag) thought it advisable to aspirate the right pleural cavity. Seven hundred cubic centimetres (twenty-eight ounces) of dark, thick, bloodstained fluid were withdrawn, with great relief to the patient who was subsequently given hypodermic injections of morphine as required. Two days later it was found necessary to aspirate the right pleural cavity again when a further litre (thirty-five ounces) were withdrawn. At this time the patient's apex beat was displaced to 17·5 centimetres (seven and a half inches) from the mid-line in the sixth intercostal space and there was a diffuse pulsation over the whole of the præcordial area with great præcordial distress. The right side of the chest both back and front was dull to percussion. It was apparent that the patient was moribund. Stimulant treatment was given and the patient lingered till December 5, 1926, when he died.

On December 6, 1926, we were given permission by the relatives to examine the lungs and pleural cavity, but not any other portion of the body. On opening the right pleural sac fluid, obviously largely consisting of pure blood, gushed out all over the table and also oozed up through the nostrils. The right lung was seen to consist of mere shreds of tissue very degenerated and very friable. One mass was isolated about the root of the right lung and was connected with what had obviously been a big cavity at the right cardio-phrenic angle. It was submitted to the Board of Health for section with the whole of the left lung which was very contracted and pushed over the left side, studded over with what appeared to be enlarged lymphatic glands. The left lung was almost obscured by the pericardial sac which was very much displaced to the left. The cavity of the heart revealed cardiac dilatation, but little else. The Board of Health reported that sections were cut of two nodules in the lung, the

mass at the hilus and a separate piece of necrotic tissue. The tissue was largely composed of degenerated cells, but in places there were definite masses of carcinomatous cells.

### Comment.

I was induced to report this case particularly because of the comparative rarity of the condition, the rapidity of onset and course. Had the condition been tuberculous, as it appeared very likely, we could have understood more easily the hæmorrhage and rapid course. It is left for the reader to imagine what took place. Whether there was an old traumatism which predisposed to malignant disease, or whether there was an old cavity which became malignant, or whether an old patch of consolidation became malignant and then degenerated, there was little evidence to show. It was rather unfortunate that we were unable to examine the abdominal viscera.

It may be interesting to conclude with a passage from Osler on "New Growths in the Lungs": "While primary tumours are rare, secondary growths are not uncommon. Carcinoma is the most common primary form. Endotheliomata and sarcomata are less frequently met with."

Osler then describes two forms, an acute galloping pleuro-pneumonic form with a rapid course, dyspnœa, cough, asphyxia, rapid emaciation and death in from six to twelve weeks and a chronic pleuro-pulmonary carcinoma. Of the latter he describes three types, a broncho-pulmonary form, a mediastinal type and a pleuritic type.

### Acknowledgment.

I am greatly indebted to Dr. A. J. Collins, Dr. J. A. Lawson, Dr. J. J. McIntosh and Dr. Boag for permission to publish this case.

## Reviews.

### A LAYMAN AND TUBERCULOSIS.

THERE has been lately addressed to the British public and to the medical profession a strange book entitled "Why Tuberculosis Exists."[1]

It consists of diverse elements including consular reports, newspaper editorials and correspondence, testimonials and comments, strung loosely together with little attempt to weld them into a homogeneous whole. The matter is broken up by startling headings. The publishers have provided a gaudy jacket with an unnecessary note of interrogation in bright scarlet and a puff designed to assist, we hesitate to say to mislead, the unwary reviewer.

The author is an architect who appears to have retired from practice and thrown in his lot with his hero Monsieur Henri Spahlinger. He makes extraordinary allegations of hindrance to the publication of his gospel and of obstruction by persons in high places, notably the British Ministry of Health and the Australian Minister for Health.

Mr. Lovell appears to be well read and capable, but as must happen to any layman writing on a medical subject, he stumbles into grotesque errors; thus he uses "T.B." for *Bacillus tuberculosis*, for pulmonary tuberculosis and for tubercular; he speaks of an "emaciation of 7 lbs.," "h'ge" in tuberculosis of glands possibly meaning hæmoptysis, "dysp." apparently for dyspepsia and of the "ganglions" of the neck, doubtless a mistranslation for glands. There are several other irritating mistranslations of French and German words, several repetitions and several inconsistencies such as 10,000 deaths a week amounting to 1,000,000 a year, as the claim that a specific remedy actually exists and the admission that it cannot be supplied, and the statement that a full exposition of the methods has been made and the admission that they remain secret.

About half-way through the book we find a description of Monsieur Spahlinger's methods. Here the author informs us that he has separated out twenty-two different toxins from cultures of the tubercle bacillus and that he employs antitoxins generated in cattle in response to injections of toxin to treat the severest forms of infection, that these antitoxins require three or four years for their preparation and that after the disease has been arrested by their use, the patient is further treated with antigens so as to induce a specific, active immunity. He does not give details of the preparation of the toxins nor of the antitoxins nor of their employment. He makes bald assertions of results achieved, but supplies no evidence in support.

Mr. Lovell reprints a number of case reports, some by well known physicians as Dr. Stephani and Dr. Leonard Williams, which claim good results extending over periods of several years, even eight or ten in some cases. They give the impression that they are genuine and reliable, but none indicate greater success than other physicians have achieved with well known remedies and it is even admitted that some of the patients had improved under tuberculin before they submitted to Monsieur Spahlinger's treatment. Some, however, protest too much, thus we frequently read that patients in advanced consumption have been restored to "perfect" health and some of them even to a more youthful appearance.

Finally, although Mr. Lovell claims that no charge is made for treatment, yet it is available only for rich patients who are prepared to make handsome donations. He admits that he himself is financially interested in Monsieur Spahlinger's scheme and that they require "cash in large amounts," £105,000 or more; they returned a donation by the British Red Cross Society of £10,000 as insufficient.

[1] "Why Tuberculosis Exists. How it may be and has been Cured and Prevented: A Book of Facts," by R. Goulburn Lovell; 1926. London: John Bale, Sons and Danielsson, Limited. Royal 8vo., pp. 220, with illustrations. Price: 6s. net.

### AUTHORITATIVE DERMATOLOGY.

DR. JAMES H. SEQUEIRA in the fourth edition of his well known textbook on "Diseases of the Skin" has given to the profession and to dermatologists in particular the best and most up-to-date work on this subject.[1]

The new edition of six hundred and forty-four pages is the same size as the previous edition, but it has been completely revised and by the rearrangement of type much new matter has been added. There are fifty-six coloured plates and three hundred and nine other illustrations, all excellent reproductions which clearly show the disease or lesion referred to in the text, a very difficult matter with such minute changes as often occur in skin diseases. The illustrations of the various types of syphilitic lesions deserve special mention.

In the new edition much has been added to the chapter on tropical diseases and in keeping with the rest of the book these are excellently illustrated.

Medical referees will find the section on occupational diseases of the skin most helpful. Cutaneous cancer and its treatment by radium and the chapters on microbic diseases of the skin are very full.

We do not know of any better book of its kind and we congratulate Dr. Sequeira on this revision of his excellent textbook which shows the evidence of his vast personal experience.

### CLINICAL CONSIDERATIONS.

AFTER the lapse of thirty-five years the fourth edition of "Finlayson's Clinical Manual for the Study of Medical Cases" has been issued under the editorship of Dr. L. Findlay and Professors Cathcart and Browning.[2] This revised edition was expressly issued in view of the present day tendency to lay greater stress on the laboratory aspect of medicine at the expense of its general principles and clinical observation which was such a prominent feature of "Finlayson" in years gone by. That out of the three

[1] "Diseases of the Skin," by James H. Sequeira, M.D. (London), F.R.C.S. (England); Fourth Edition; 1927. London: J. and A. Churchill. Royal 8vo., pp. 656, with illustrations. Price: £2 2s. net.

[2] "Finlayson's Clinical Manual for the Study of Medical Cases," Edited by Carl H. Browning, M.D., E. P. Cathcart, M.D., F.R.S., and Leonard Findlay, M.D., D.Sc.; Fourth Edition; Revised and Augmented throughout by various contributors; 1926. London: G. Bell and Sons, Limited. Post 8vo., pp. 830, with illustrations. Price: 18s.

editors only one is a clinician (Dr. Findlay) while the other two are prominent exponents of laboratory methods, makes the appearance of this standard book all the more remarkable. Besides the editors all the chapters have been revised by twenty contributors. The only chapter which is retained in its entirety is the first by Sir W. T. Gairdner on "The Physiognomy of Disease." Gairdner's knowledge of disease, powers of observation and impressive teaching left a lasting stamp on all those who came under his influence, so much is this so that after many years this quiet, cultured gentleman still remains in the minds of those who knew him with affection and veneration. All the other chapters are practically rewritten and a new one is added by Professor Cathcart on the ductless glands.

As the first edition was the pioneer in ward work and proved to be the mentor of generations of students we feel satisfied the fourth edition will be an unfailing guide in the path of clinical righteousness to many more.

## RECENT WORK IN MEDICINE.

The editors of the volume on general medicine in the Practical Medicine Series for 1926 are to be congratulated on having coped successfully with the difficult problem of summarizing the progress made during the previous twelve months.[1] The articles quoted are for the most part well chosen, though occasionally there is a tendency to overload a subject with a plethora of supporting statements and difficulty has been experienced in avoiding repetition of well established facts. Where important conclusions are concerned this latter trend is of service. For example "the functional capacity of the heart is of more importance than the physical signs" and "enlargement of the spleen during the treatment of cardiac disease is often an evidence of recurrent endocarditis." The editorial comments are excellent and serve to control unjustified conclusions, in regard to which enthusiasm has outrun judgement. Typical examples of such are: "Rheumatism is preventible by the eradication of local infections" and "subacute bacterial endocarditis results invariably from the *streptococcus viridans*."

In the section on infectious diseases a complete summary is presented of the means of eliciting susceptibility and establishing immunity in those with deficient natural protection. With full cooperation between public health authorities and private practitioners, diphtheria should be an eradicable disease. Intraperitoneal injection of antitoxin is recommended when speedy effect is desired with a minimum of shock.

The use of ether in pertussis is growing in favour and the adoption of the rectal route is of service when intramuscular injection presents difficulty. Diathermy has been found of value in the testicular metastasis of mumps and in pneumonia. In enteric fever fear of giving solid food is abating and the results of generous feeding are distinctly encouraging. The subject of organotherapy is dealt with intelligently and Lisser's summary of the present achievements and future prospects is quoted *in extenso*. Lisser divides the therapeutical indications into substitional (supplying deficient gland secretion) and pharmacodynamic. He deprecates the use of pluriglandular preparations. Thyreoid preparations have been found useful in the irregular and profuse early menstruation of young girls and anterior pituitary lobe in that form of post-adolescent hypopituitarism with girdle adiposity and scanty irregular menstruation in women between twenty and twenty-five years of age.

Calmette's results are given of treatment by inoculation of infants with the Calmette-Guérin bacillus to protect against tuberculous infection and they are sufficiently encouraging to warrant more extensive trial. Tuberculosis occupies the major portion of the section on diseases of the chest and valuable papers on its pathology, diagnosis and treatment are quoted. The consensus of opinion is in favour of earlier resort to the production of artificial pneumothorax in progressive unilateral tuberculosis and severe hæmoptysis. Contraindications are diffuse activity in the other lung, cardiac embarrassment or tuberculosis of other organs. The use of pneumococcal vaccine as a prophylactic and of the serum as a curative measure appears to be gaining favour.

The subject of hæmatology receives less consideration than it should. Earlier and repeated transfusion of blood is recommended and attention is drawn to the importance of differentiating between acute lymphatic leuchæmia and an acute infective lymphoblastosis, the prognosis of the two conditions being exactly opposite. Eddy and Downs strongly recommend the use of dried powdered spleen and red bone marrow in secondary anæmia. The need for the adoption of sanatorium methods in the treatment of heart disease is emphasized and most practitioners will cordially agree with this conclusion. That the presence of pericarditis is frequently missed will be generally conceded and attention is drawn to the increased mobility of the cardiac dulness and the cardiac shadow. Barium chloride has been of service given orally in two gramme doses thrice daily. The use and risks of quinidine are well set out in a paper by Riecker and caffeine sodium benzoate is recommended for cardiac collapse to be given intravenously in sixty-five to one hundred and thirty milligramme doses.

In the section on diseases of the stomach the present position of treatment of gastric ulcer is discussed, the tendency being towards conservative methods. A valuable contribution is that by Orr and Haden on the hypodermic or subcutaneous use of sodium chloride solution in intestinal obstruction as an adjuvant to operation. The intraperitoneal injection of oxygen in cases of tuberculous peritonitis and the diagnostic use of "Novoprotein" injections for suspected chronic appendicitis are among the suggestions contained in this section. The ætiology of gall stones is dealt with by Aschoff and the use of the bromine and iodine salts of tetraphenolphthalein for cholecystography is regarded as well established. The volume is well printed, is of handy size and is likely to be of distinct value to physicians either in general or special practice.

[1] "The Practical Medicine Series, Comprising Eight Volumes on the Year's Progress in Medicine and Surgery." Under the General Editorial Charge of Charles L. Mix, A.M., M.D.; General Medicine; 1926. Chicago: The Year Book Publishers. Crown 8vo., pp. 737, with illustrations. Price: $3.00 net.

## ULTRA-VIOLET RAYS IN GENERAL PRACTICE.

A volume on the use of ultra-violet rays in general practice is the result of twelve months experience in his own general practice by W. A. Troup.[1] Though this experience does not warrant the production of a book on the subject, the result is quite a useful summary of the main points to be mastered by one beginning to use this method in treatment. In its fifty-three pages there is of course room for no more than a summary. Historical, physical and general considerations including descriptions of various lamps *et cetera*, occupy thirty of these pages, the remaining twenty-three containing descriptions of therapeutic application. Some of the statements here do not carry conviction, particularly with the knowledge that there is only one year's experience behind them. For instance under the heading of "Conditions Cured or Alleviated by Ultra-violet Rays which do not Respond readily to other Forms of Treatment" is this terse statement: *Psoriasis*—this disease can invariably be cured!

There are other equally sweeping statements which we cannot help feeling that the author will modify with even one more year's experience. This is particularly the case when reports from workers with vastly greater experience are so much more guarded. Over-sanguine statements such as these tend to throw suspicion on a method which is proving of really great value with many workers.

[1] "Ultra-Violet Rays in General Practice," by W. Annandale Troup, M.C., M.B., Ch.B. (St. Andrews); 1926. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 71, with illustrations. Price: 4s. 6d. net.

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## Resolutions of Congress.

Under the old order of things the final plenary session of the Australasian Medical Congress passed resolutions which if they had been translated into action, would have had far reaching results. These proposals were moved by members at sectional meetings and no doubt were usually the result of much thought and an earnest desire to remedy some defect in the relationship between the medical profession and the public or to initiate some movement for the improvement of the public health. The motions usually received consideration by a handful of members present at the sectional meetings. At the larger plenary session the majority of those present had no special knowledge of the circumstances surrounding the proposals. If a discussion took place, it was usually conducted by the few interested members from the particular section at which the motion had emanated. Had these proposals been referred to the following session of Congress, so that the whole profession would have an opportunity of studying the problems involved, the opinions expressed by resolution of the majority of those attending a plenary session might have been valuable. As this was not done, the so-called resolutions of Congress were usually adopted without thought by tired practitioners anxious to secure some leisure after an arduous week of medical meetings.

When the Federal Committee of the British Medical Association in Australia reorganized the Congress and drew up the regulations in 1921, special precautions were taken to remedy this unsatisfactory state of affairs. It will be remembered that regulation thirteen, which deals with sectional meetings, contains a subsection (*f*) to the following effect:

> Resolutions of sectional meetings: If it appears to any Section that action should be taken in connexion with any matter which had been the subject of discussion at its meeting, any proposal for such action shall be referred to the Federal Committee and shall not be published in the lay press.

This regulation prevents the submission to a general meeting of any resolution carried at a sectional meeting. It provides for a careful inquiry by the Federal Committee and if desirable by the Branches of the British Medical Association in Australia and New Zealand into the subject matter of the proposals. It leaves the Federal Committee a free hand to take any action that may be deemed necessary after this inquiry. There are therefore no longer any resolutions of Congress, opinions ostensibly carrying the endorsement of the whole medical profession of Australia.

In actual practice the filter through which proposals adopted at sectional meetings must pass, is more efficient than the wording of regulation would suggest. At the second session in Dunedin eleven proposals were made at sectional meetings and passed as resolutions. These resolutions were remitted to the Executive Committee. There was a final general meeting on the last day of the session and at this meeting the resolutions were read by the Honorary General Secretary. Dr. L. E. Barnett, the President, explained that the meeting had no power to accept or refuse any of the proposals. He invited those present to express their opinions on the several matters dealt with in the resolutions, in order to assist the Executive Committee and the Federal Committee in determining what action if any should be taken. No discussion took place; the first filtration thus removed no sediment. The next step was for the Executive Committee to consider the proposals and to arrive at a decision whether any or all of them should be referred to the Federal Committee. The second step will probably prove valuable to the Federal Committee when it meets on April 27, 1927, and considers these matters. Some of the proposals have general application and will probably be referred to the several Branches of the British Medical Association for consideration and report before any action is adopted. Others have limited application and will no doubt be referred to that Branch of the Association concerned with the subject matter. It may be that some of the proposals will be regarded as im-

practicable. The slow moving, thorough machine of the Federal Committee acts as an admirable brake on the excessive zeal of impetuous reformers at sessions of Congress. In the course of time the profession will learn that it is not worth while to suggest the creation of ponderous bodies to carry out functions that could well be undertaken by single individuals or to ask for the promulgation of considered opinions of the medical profession that may become embarrassing in the course of a few years. On the other hand useful reforms may wisely be initiated in this manner.

## Current Comment.

### COLITIS.

A diagnosis of colitis is frequently made without justification. The presence of diarrhœa and a history of "blood and slime" in the stool, perhaps with some pain and tenesmus, are often regarded as sufficient to justify the use of the term colitis. The patient's description of blood and mucus is accepted without careful investigation. It is unfortunate that there is a tendency among medical practitioners to use specific terms to describe conditions which bear superficial resemblance only to those indicated. Most practitioners have used the word influenza to describe a febricula caused by the *Micrococcus catarrhalis* or allied organism and such inaccurate diagnoses as dyspepsia, rheumatism and heart strain are a matter of every day occurrence. Occasionally in matters of this sort an accurate diagnosis may be impossible; sometimes the want of accuracy indicates lack of knowledge, but more often carelessness is responsible. As a rule no serious harm results to a patient when a wrong diagnosis of colitis is made. On the other hand, diarrhœa and blood in the stool may be the first indications of the presence of malignant disease. Colitis often occurs as part of a general catarrhal inflammation of the intestinal tract and it may be seen as an accompaniment of organic disease of the large bowel. It is also a manifestation of dysenteric infection. At the same time simple colitis occurs occasionally as an independent disease.

It will thus be seen that it is necessary for the adequate treatment of colitis that a clear conception be obtained of what colitis actually is, what its manifestations are and what means are available for its recognition. These points were brought out in a clear way at a recent discussion held by the Royal Society of Medicine.[1]

In opening the discussion A. F. Hurst pointed out that the patient's statement in regard to the character of the stools should not be accepted, but that the stools should be examined. In his opinion natural stools (not those obtained after use of an aperient or an enema) should be examined by the clinician and should be submitted to microscopical and bacteriological examination. The presence of mucus in the stools is not evidence of mucous colitis; every healthy mucous membrane secretes mucus. For this reason the patient should be subjected to sigmoidoscopy without any preparation by aperients or lavage. In true colitis the mucus always contains pus cells. Obvious blood in a stool with mucus and pus denotes ulceration, but smaller quantities, recognizable only with the microscope, may be present in acute colitis without actual ulceration. When the source of blood and pus in the stool is the inflamed mucous membrane, the stool is always soft and generally liquid. Hurst holds that colitis of all kinds is almost always due to infection of the mucous membrane of the colon. He refers to the suggestion of Gemmel that idiopathic ulcerative colitis is due to dysentery. He also draws attention to the fact that the appearance of the colon as seen with the sigmoidoscope is similar in ulcerative colitis to that seen in bacillary dysentery. He has not been able, however, to isolate the organism from swabbings and he has not been able to produce agglutination of known strains of *Bacillus dysenteriæ* by the patient's blood.

Much work has been done in Australia on the subject of intestinal infections. Little has shown that the bacillus of dysentery is responsible for the condition of many infants whose stools contain blood and mucus and who suffer from tenesmus, fever and collapse. Patterson and Williams, working in Melbourne in 1920-1921, found that pathogenic organisms could be cultured from the stools of 71% of patients passing blood and mucus or muco-pus; of these organisms 66·6% were Flexner dysentery bacilli. In view of the fact that dysentery is present in such a high proportion of cases, the importance of distinguishing between amœbic and bacillary dysentery is evident. Amœbic dysentery is occasionally seen in Australia. Infections have been discovered in returned soldiers and in travellers from other parts of the world and Nelson and Shearman have reported an instance in which amœbic dysentery was apparently contracted in Australia. It is here that stress may be laid on Hurst's advocacy for the use of the sigmoidoscope whenever disease of the colon is suspected. He points out that ulcerative colitis whether due to dysentery bacilli or not, can be recognized at a glance. On the other hand, amœbic dysentery presents quite a distinctive picture. Ulcerative colitis originates in the most distal segment of the colon and persists there longer than in any other part. When, therefore, stools containing blood and pus are passed by patients in whom the sigmoidoscope has revealed no evidence of ulcerative colitis, a growth of the colon is almost certainly present. If a growth is situated within twenty-five centimetres of the anus, it should be discovered by use of the sigmoidoscope.

Reference should be made to the analogy drawn by Hurst between muco-membranous colitis and asthma. In asthma over-activity of the vagus leads to spasm of the bronchial muscles and to excessive

[1] *Proceedings of the Royal Society of Medicine*, February, 1927.

secretionof mucus. Owing to the spasm the mucus is often retained long enough for it to undergo coagulation. In uncomplicated cases there is no bronchitis. In the colon the spasm and over secretion of mucus are due to similar nervous influences; the mucus is retained owing to the spasm and is coagulated by a ferment, mucinase, present both in the bronchial and intestinal mucous membrane. Just as asthma may be complicated by bronchitis as a result of treatment by irritating inhalations, so muco-membranous colic, as it should be called, may be complicated by a mild form of true colitis as a result of injudicious local treatment.

In view of the bacteriological findings and of the appearance of the mucosa described by Hurst, it is not surprising that he advocates the general use of antidysenteric serum in all cases of ulcerative colitis. He has found that as a rule this form of therapy yields excellent results, but occasionally it has been a complete failure. He realizes that the treatment may be non-specific and nothing more than a form of protein shock. L. S. Dudgeon said that he could see no adequate explanation for the results obtained by Hurst. In his opinion antidysenteric serum is of use for combating the toxæmia of acute bacillary dysentery, but is useless for the treatment of the intestinal ulceration. Thus, even if ulcerative colitis is regarded as a form of true bacillary dysentery, the use of antidysenteric serum will not explain the successful results. The suggestion that the action of the serum is not specific and that the result is due to protein shock, cannot be accepted, because Hurst failed to obtain success with horse serum.

Although several speakers referred to the immediate relief of symptoms following the use of emetin in amœbic dysentery, no reference was made to the danger of using this drug in bacillary infection. For this reason every available means must be used to secure a correct diagnosis. In this connexion Hurst's remarks regarding the use of the sigmoidoscope may again be emphasized. If this discussion does no more than lead to a wider use of the sigmoidoscope, good will be accomplished.

## THE ISLANDS OF LANGERHANS IN THE HUMAN PANCREAS.

The study of microscopical anatomy is indispensable to the understanding of normal physiology in the same way that knowledge of the normal must precede any attempt to cope with the pathological. In other words structure and function are interdependent. Attempts to describe the cold facts revealed by histological examination are not always welcomed by the clinician who can see in them little application to the problems before him. The application is there, however, and if sought diligently it will usually be found.

The structure of the pancreas has been described by many authors and a great deal that is of importance has been learned. In 1924 Gordon Cameron published in this journal some important observations on the *post mortem* changes in the pancreas. He devoted particular attention to the intracellular granules and pointed out that the early *post mortem* changes in the islands of Langerhans were similar to those occurring in experimental and human diabetes. The relationship of the islands of Langerhans to the surrounding structures has given rise to much discussion. It is claimed by some investigators that the islands possess a limiting capsule which cuts them off from the remainder of the pancreatic tissue. Other workers hold that the islands are directly continuous with the surrounding acini and others again describe communications from the islands to the pancreatic ducts. Sadao Otani has recently investigated this relationship.[1] He removed the pancreas from the bodies of forty-six persons ranging in age from three days to seventy-six years. In all cases the organ was removed from the body within five hours of death. Otani found that when suitable silver stains were used the acinus was surrounded by fine black fibres which could be recognized as a lattice work in serial sections. These fibres were seen to serve as the basement membrane of the acinar epithelium and as the supporting framework of the blood capillaries. The fibres appeared between the acini and the islands. Otani recognized them as the basement membrane of the acini Branches of the fibres extended into the islands and surrounded the insular capillaries. Otani describes islands of three types: islands continuous with acini, islands directly continuous with ducts and islands separated from the surrounding tissues. Islands continuous with acini were the most frequent, the other types of island were found only after considerable search. The three different types occurred occasionally in the same pancreas more or less in the same proportions. These findings differ from those of other observers. Otani attributes this difference to the method employed and to the number of specimens examined. He holds that his findings are not influenced by pathological changes in the organ, because the specimens were taken from persons of all ages and because similar appearances were found in healthy animals. On the other hand, islands continuous with acini were predominant in new-born babies and Otani takes this as indicating that the structure of the pancreas undergoes further changes in the postfœtal period. In view of his acceptance of the limiting fibres around the islands as the basement membrane, he concludes that the occurrence of a thick, collaginous capsule is proof of a pathological condition of the pancreas.

It is obvious that apparently isolated islands will have capillary communication with the surrounding tissue. The islands are the source of the pancreatic hormone. If the black fibres described by Otani constitute the basement membrane of the acinar epithelium and the supporting framework of the capillaries, the cutting off of the islets must be regarded as a pathological process. Otani describes this as a regular occurrence in advancing age. It would seem more probable that this is an early fibrosis and therefore not a physiological process.

[1] *The American Journal of Pathology*, January, 1927.

# Abstracts from Current Medical Literature.

## OPHTHALMOLOGY.

### Sympathetic Panophthalmitis.

W. E. Shahan (*American Journal of Ophthalmology*, February, 1927) relates the history of a man who was injured in the left eye by a nail. When seen eleven days later by the author, the patient presented a large suppurating wound in the ciliary region, a yellow reflex from the vitreous, chemosis and exophthalmos. Reasoning that the panophthalmitis was prophylactic against sympathetic involvement of the other eye, the author performed evisceration. Twenty days later inflammatory signs appeared in the right eye. In spite of the fact that the stump of the left eye was enucleated, the right eye became worse—a definite sympathetic ophthalmitis. Treatment with atropine and salicylate of sodium and "Neosalvarsan" resulted in a favourable issue.

### Modified Lagrange Operation.

Joel Whitaker (*American Journal of Ophthalmology*, February, 1927) in performing the Lagrange operation makes two radial incisions in the conjunctiva starting from the limbus at the ten o'clock and the two o'clock positions. The conjunctiva is separated from the limbus and dissected upwards. An incision is made with a keratome well back in the limbus and the corneal tip is grasped with a rat-tooth forceps and cut off with scissors. The iridectomy flap is then performed. The conjunctival flap is then sutured on each side to the conjunctiva at the nine o'clock and the three o'clock positions.

### Visual Pseudohallucinations after Encephalitis Lethargica.

C. Pascheff (*Revue Générale d'Ophthalmolœgie*, August, 1926) has already described among the late manifestations of *encephalitis lethargica* a special tic or syndrome of periodical deviation of the eyes upwards. This is associated with wide opening of the lids, elevation of the eyebrows, wrinkling of the forehead and a fixed look into space as if the patient were fixing an imaginary object. The case is reported of a woman of twenty-six whose illness began five years previously. She was confined to bed with rigidity of the extremities. She had these ocular crises from two to seven times a day. She described the "visions," she saw men and women talking, a train, a she-bear and other things. She quite realized they were only "visions."

### Diphtheria Antitoxin for Sympathetic Uveitis.

F. H. Verhoeff (*Archives of Ophthalmology*, January, 1927) claims remarkable results from the injection of diphtheria antitoxin in cases of sympathetic uveitis. A concentrated form was employed, 20,000 units being contained in fifteen cubic centimetres of fluid. The details of seven cases are given. Recovery was more prompt and complete when the treatment was begun early. In four of the cases normal vision was finally restored. Failure in one case was due to the long delay nine weeks before treatment. Contrary to usual experience operations could safely be performed on the affected eye while the patient was under the influence of antitoxin. The patient is tested for sensitiveness to antitoxin. If he is not sensitive, 20,000 units are heated to body temperature and injected. The same dose is given daily for one week. If then the ocular congestion subsides, the dose is given every week; if not, daily injections are continued until improvement is definite or symptoms of anaphylaxis appear. If the patient is sensitive or later on anaphylactic, the antitoxin is given in divided doses.

### Treatment of Glaucoma with "Glaucosan" and "Amine-Glaucosan."

C. Hamburger (*Archives of Ophthalmology*, November, 1926) found that subconjunctival injection of 0·5 cubic centimetre of a one in a thousand solution of suprarenin had a good effect on *glaucoma simplex*. Vision and fields could be preserved for years when miotics had lost their effect. After suprarenin treatment miotics frequently regained their effect. To obviate the danger of increased blood pressure from the injection of suprarenin a mixture was made of lævogyrate suprarenin solution one in five hundred, with dextrogyrate suprarenin one in one hundred. A highly concentrated solution of lævogyrate suprarenin salts, called "Glaucosan" drops did not increase the blood pressure when instilled into the eye. This becomes marble white, the pupil dilates and the intraocular tension falls. This condition remains for several days, sometimes even for weeks. Two drops are instilled, the eye being moved about so that the drops get into close touch with every part of the eye. This is repeated three to five times at intervals of a quarter of an hour. The drops are put up in tubes in small quantities sufficient for one treatment. "Glaucosan" drops should be used when miotics lose their power or are not tolerated. The most powerful miotic is "Amine-Glaucosan," its chemical name being $\beta$ imidazolyæthyl-amin contained in *secale cornutum*. This may be injected under the conjunctiva, but is painful; stronger solutions may be used as drops. "Glaucosan" drops must not be injected.

### Hemangioma of the Chorioid.

E. J. Lent and M. B. Lyon describe a case of hemangioma of the chorioid, the twenty-sixth reported (*The Journal of the Indiana State Medical Association*, November, 1926). This condition is of congenital origin and nearly half the patients have had nævi elsewhere on the body. Eleven of them were between eleven and twenty years of age. The position of the growth is always near the nerve head. In the authors' case examination revealed a bluish-white bulging without pigmentation in the lower nasal quadrant, the size and shape of a split pea. The eye was removed after a diagnosis of sarcoma had been made. If left, the growth causes detachment of the retina and glaucoma and bone formation in the chorioid. These growths are not malignant and if a true diagnosis be made the eye need not be enucleated until painful glaucoma occurs.

### Plain Glass and Prism Test for Malingering.

E. H. Schild (*American Journal of Ophthalmology*, October, 1926) describes the application of a piece of plate glass with parallel sides bevelled at one end to form a prism as a good test for suspected monocular malingering. With the prism end held uppermost before the good eye and the plain glass before the pupil there is no effect; by lowering the glass a trifle enough to bring the dividing line midway across the pupil the effect of a double prism is produced and the eye sees a double image. Again lowering the glass the effect of a single prism is produced. Suppose the left eye is under suspicion, hold the prism in front of the right eye; in the first position there is no difference; in the second position there will be two images in the right eye and one in the left. This may fuse with the right image and the patient may say that he sees two images. Now unknown to the patient move the glass down to the third position (this is the critical part of the test) and ask how many he sees. If the left eye is blind he will see only one, but if he says two, his left eye is not blind A horizontal row of letters of different sizes may be substituted for an object. With the glass in the third position ask the patient to read the lower row of letters. If he does so it will give roughly the visual acuity of his so-called blind eye.

## LARYNGOLOGY AND OTOLOGY.

### Thymic Death in an Adult.

David N. Husik (*The Atlantic Medical Journal*, September, 1926) reports the death of an apparently healthy adult patient during the operation of tonsillectomy, from a condition of *status lymphaticus*. The patient, a female, aged twenty years, had had two attacks of joint inflammation during the course of which acute tonsillitis occurred. Her previous history was clear except for recurrent sore throats and bronchitis in childhood. Eight milligrammes (one-sixth of a grain) of morphine and 0·43 milligramme (one one-hundred and fiftieth of a grain) of atropine sulphate were given hypodermically half an hour before operation. Four

cubic centimetres of an 0·5% solution of "Novocain" to which 0·12 mils (two minims) of a one in a thousand solution of adrenalin hydrochloride had been added, were injected into the tonsillar area after the fauces had been painted with a 10% solution of cocaine. Enucleation of the left tonsil was immediately commenced and completed with great difficulty. One and a half minutes after the injection of "Novocain," the patient became very excited, then cyanotic, spastic and unconscious. Death occurred in spite of cardiac stimulation in four minutes. *Post mortem* a large thymus gland was found with hypoplasia of the heart and arteries and chronic generalized lymphadenitis. Since this fatality the author has had an X ray examination of the chest made in all patients requiring tonsillectomy. All children up to fourteen years of age recommended for removal of tonsils and adenoids at the Massachusetts General Hospital, are also examined in this way. All patients with a broad superior mediastinum are given four X ray treatments of one-third of an erythema dose at intervals of ten days before operation is undertaken. Of 2,344 children, 7·5% manifested a thymus shadow and of these 60% have been operated upon successfully after X ray treatment.

### Paracusis.

MR. VERN O. KNUDSEN and ISAAC H. JONES (*The Laryngoscope*, September, 1926) have carried out investigations on the hearing of patients suffering from paracusis. This has been a subject of controversy for one hundred and fifty years, since Willis published his findings. The authors hope that apart from the significance in prognosis a better understanding of paracusis may lead to a better conception of the physiology of the cochlea. Experiments were carried out on one hundred and eighty-seven consecutive patients, complaining of deafness. The facts to be determined were whether paracutic patients hear better in a noise and if so, why. Hearing tests were carried out by means of the "audio-amplifier," an electrical instrument by which exact findings can be registered. The one hundred and eighty-seven patients were classified as follows: seventy-three suffered from perceptive deafness, fifty-eight suffered from mixed perceptive and conductive deafness and fifty-six from deafness purely conductive or almost entirely conductive. None of the patients with perceptive deafness exhibited paracusis, while 25% of the mixed type and 46% of the conductive type showed this condition. Of the above ten paracutics, two perceptives and three persons with normal hearing were further tested. Investigations were carried out to determine the interfering effect of noise upon the acuity of hearing of the pure tones, to determine the effect of noise upon speech sounds and to determine the effect of pure tones on speech sounds. The noise apparatus was devised to act as a "blanket," containing all of the audible frequency components. To this end an electric circuit of vacuum tube oscillators with an electrically driven rotary condenser, was attached to a specially constructed telephone receiver which enabled the noise to be added directly in conjunction with the speech or pure tone tests being carried out. Hearing of the pure tones was audiometrically measured in the quiet; then the tests were repeated in the presence of the noise which was controlled from barely audible loudness up to a magnification of one thousand times. The speech articulation tests were similar to those evolved by telephone engineers in testing electrical equipment. These tests were carried out in the quiet, with the noise amplifier functioning at different intensities and finally with the introduction of low, medium and high tones of different intensities. The findings of these experiments are all indicated in graph form and prove that for hearing pure tones in the presence of noise, the hearing of a paracutic is equally impaired with that of the normal person, while the perceptive was less affected as the noise increased. For hearing speech in the presence of noise it was found that as the noise increased, the percentage of judging articulation decreased in all three classes; but in the presence of a very loud noise the paracutics showed a higher appreciation for vowel sounds than either of the other two classes. For hearing speech in the presence of pure tones it was found that the tones of all pitches interfered with hearing of speech. The interference by low tones was greater than by high tones, the low pitch tones interfering with both vowels and consonants, the high pitched tones particularly with consonants. Qualitative tests were also carried out. The authors finally summarize the opinions of many previous investigators of this subject and add their own conclusions which are that a paracutic does not hear better in a noise, but has an advantage over the normal or perceptively deaf person. This is explained by his being less disturbed by the noise. Paracutics hear low tones with difficulty and tones of 1,000 D.V.'s to 2,000 D.V.'s best, while hearing above this varies in different patients. As noises mostly met with are of low frequencies, a paracutic is less affected by these than the normal or perceptively deaf person. Further, persons in the other two classes being more affected, tend unconsciously to raise their voices in the presence of a noise.

### Iodized Oil as an Aid in the Diagnosis of Chronic Sinusitis and of Maxillary Cysts.

PAUL B. MACCREADY (*The Boston Medical and Surgical Journal*, September 2, 1926) has found the injection of iodized oil into the nasal sinuses an aid to diagnosis of sinusitis in conjunction with X ray examination. The author uses a 10% solution of metallic iodine in oil of poppy seeds and finds this to be of sufficient strength for these examinations. The cavity to be examined is first irrigated with normal saline solution and thoroughly drained, as iodized oil is insoluble in water. The iodized oil is then injected and an X ray examination immediately made, stereoscopic views anteriorly and laterally being taken. One patient who was normal on clinical examination and washing out of the antrum, manifested after injection of iodized oil an antrum cavity filled by polypoid hypertrophy of the lining membranes. A dentigerous cyst after injection was clearly defined and localized, giving the surgeon useful information as to extent before operation. The author claims that thickening of the walls of the antral cavity in different areas is often indicative of the source of infection. For example, if a tooth is responsible, the antral floor is thickened; if the ethmoids are at fault, the superomedial wall is thickened. The oil is quickly removed from the sinus and thus has little therapeutic value. No ill effects have been noticed following injection, but "temporary congestion of the cervical glands" has been encountered.

### Cystic Serous Meningitis.

G. J. JENKINS (*The Proceedings of the Royal Society of Medicine*, Section of Otology, September, 1926) discusses cystic serous meningitis and describes the condition of two patients dealt with by operation. Both had had long standing ear sepsis and a radical mastoid operation had been performed on the affected side some time prior to the onset of symptoms. The symptoms complained of were vertigo, headache on the affected side and occipital regions and tinnitus. Clinical examination over a period of months in each case revealed finally a spontaneous nystagmus, past pointing to the affected side, slight incoordination of the movements of the upper extremity of the affected side, while hearing tests revealed no cochlear lesion. The Wassermann test yielded no reaction and the cerebro-spinal fluid was normal in each instance. In addition one patient had severe indefinite symptoms which were at first thought to be due to hysteria with Rombergism to the affected side, whilst the other had papillœdema of both eyes. Treatment was identical in each case. The *dura mater* was exposed between the lateral sinus and the labyrinth and when it was opened, a gush of cerebrospinal fluid at once occurred, while a spatula entered an apparently large space. This space was drained by iodoform gauze left *in situ* for one week. Relief of symptoms was definite and speedy, both patients recovering the function of the arm and losing all symptoms of vertigo, headache and tinnitus. Several members in discussing this subject, described similar cases and considered that the condition simulated cerebellar lesions, while being more amenable to treatment. The lesion is due to a shutting off of the subarachnoid space during a local meningitis, but usually no organisms are present in the fluid.

# British Medical Association News.

## SCIENTIFIC.

A meeting of the Victorian Branch of the British Medical Association in Australia was held in the Medical Society Hall, Albert Street, East Melbourne, on March 2, 1927, Dr. R. J. Bull, the President, in the chair.

### Anæsthesia and the Newer Anæsthetics.

Dr. Reginald Howden read a paper entitled: "Anæsthesia and the Newer Anæsthetics: Ether" (see page 596).

Dr. F. W. Green read a paper entitled: "Anæsthesia and the Newer Anæsthetics: The Intratracheal Administration of Ether" (see page 598).

Dr. W. S. Newton read a paper entitled: "Anæsthesia and the Newer Anæsthetics: Nitrous Oxide and Oxygen" (see page 599).

Dr. G. L. Lillies read a paper entitled :"Anæsthesia and the Newer Anæsthetics: Ethylene and Oxygen" (see page 601).

Dr. W. L. Carrington said that he had been very much interested by the papers presented and particularly by those dealing with nitrous oxide and ethylene anæsthesia.

He had been very greatly impressed with the advantages of nitrous oxide as an anæsthetic agent during his term as casualty surgeon at the Melbourne Hospital in 1923. Although the apparatus then available was rough in that it was not provided with a reducing valve and failed in the accurate control of the proportions of the gases administered, it was used in all sorts of emergencies and justified itself again and again.

The advantages of anæsthesia induced by the "safety" gas and oxygen machine of the type exhibited by Dr. Newton over ether anæsthesia were well exemplified in the operation for the relief of exophthalmic goître. The difference in favour of nitrous oxide and oxygen was amazing. The anxiety inseparable from this operation under ether anæsthesia was minimized by the use of nitrous oxide and oxygen. Not only was the patient's condition very much better during the operation, but convalescence was more rapid. He regarded nitrous oxide and oxygen anæsthesia as a factor almost equal in importance to preoperative iodine treatment in the surgery of exophthalmic goître.

In very bad emergency surgery the results attending the use of nitrous oxide and ethylene were very much better than those obtained with open ether. The newer anæsthetics extended the field of the surgeon, rendering it possible for him to operate successfully under circumstances formerly regarded as hopeless. He recalled the case of a woman, aged seventy-eight years, upon whom it was necessary to operate for the relief of intestinal obstruction of seven days' duration. After the failure of local anæsthesia the patient had been successfully brought through the operation by means of nitrous oxide and oxygen. He was also reminded of a child whom rupture of the spleen had reduced to a very critical condition of exsanguination. The administration of ethylene had permitted the necessary operative measures to be carried out and the child recovered, but he was convinced that the little patient would have died had ether been administered.

When employing nitrous oxide and oxygen or ethylene, they could assure their patients that they would be none the worse and that there would be no troublesome after effects. The rapid induction and recovery from nitrous oxide and ethylene gave these anæsthetic agents a very great advantage in casualty work at a busy hospital.

All difficulties in administration arose from insufficient mastery of the apparatus and technique. The machine as designed was essentially a "one man show," in that it ought always to be used by the same person. Difficulties arose when the same machine was used by a number of individuals each of whom made different adjustments. In his opinion nitrous oxide and ethylene were the anæsthetics of the future.

Mr. Alan Newton tendered his congratulations to the readers of the papers. Ether anæsthesia had been considered very fully by Dr. Howden and Dr. Green and there appeared to be no scope for discussion.

Dr. W. S. Newton had referred to the absence of operative shock when anæsthesia was induced and sustained by nitrous oxide and oxygen. The experimental work of Dale provided interesting confirmation of this clinical observation. Dale found that the injection of histamine preparations into unanæsthetized cats did not produce the phenomena of shock. When the injection was made under nitrous oxide and oxygen anæsthesia there were likewise no manifestations of shock, but when the animals were anæsthetized with ether the injection of histamine was followed by all the evidence of shock.

There appeared to be no doubt whatever that nitrous oxide and oxygen was the most suitable anæsthetic in the presence of surgical shock.

Dr. Carrington had referred to the subject of exophthalmic goître and he could confirm from his own experience Dr. Carrington's statement that with nitrous oxide and oxygen anæsthesia the operative risk was very greatly diminished and that in general the patients were very much less upset by the operation than was the rule with ether anæsthesia. He considered intratracheal administration of ether especially a bad method in exophthalmic goître, as when given in this manner the ether seemed to occasion more respiratory troubles than attended the ordinary "open" method.

Altogether he had performed one hundred and seventeen operations for exophthalmic goître with anæsthesia supplied by the nitrous oxide and oxygen machine and in the series there had been only one death. He was sure that the single fatality was not due in any way to the anæsthetic.

His experience of ethylene as an anæsthetic was limited, but it had been conspicuously successful in the case of an old lady upon whom he was obliged to operate for gangrene of the gall bladder. He had been impressed with the efficacy of ethylene in desperate circumstances which precluded the use of ether upon other occasions also, and felt justified in advocating the more extended use of both nitrous oxide and ethylene in suitable cases.

Dr. F. L. Davies said that his unavoidably late arrival placed him at a disadvantage in contributing to the discussion on the papers.

He felt that although they should allow a special sphere of utility to nitrous oxide and oxygen and ethylene, a sphere which previous speakers had indicated, they must not be carried away by enthusiasm for the newer anæsthetics. Dr. Lillies had recorded a mortality of 3% in the use of ethylene. He submitted that an anæsthetist would have to be singularly unfortunate in being asked to anæsthetize a large number of very bad patients if he were to experience a mortality of 3%. He did not wish to be understood as saying that the new anæsthetics were not good, but his view was that they had yet to prove themselves.

He quite agreed with Mr. Alan Newton that the intratracheal method of administration of ether was not suited to exophthalmic goître; in fact, it was very bad for these patients. The introduction of the catheter required a depth of anæsthesia which was undesirable and which was unnecessary for purposes of the operation.

He urged those contemplating the use of intratracheal method of producing anæsthesia to make sure they secured a proper apparatus. He had recently been asked by a doctor to inspect an anæsthetic machine which he was considering purchasing, and had no hesitation in condemning it as not possessing the elements of a well designed apparatus. He was indignant that such a bad outfit should be offered for sale and he advised all about to purchase an intratracheal apparatus to protect themselves by consulting with someone experienced in the use of such machines.

He was interested to hear Dr. Lillies say that he did not give an injection of morphine prior to the administration of ethylene. In his opinion the preliminary injection was a large factor in slow recovery from anæsthetics. He wished to ask if preliminary injection of morphine had been observed to delay the characteristically rapid recovery from the effects of nitrous oxide and oxygen.

DR. H. HUME TURNBULL in reply to Dr. Davies's question said that as far as he had been able to observe the injection of morphine prior to nitrous oxide anæsthesia did not delay the subsequent return to consciousness.

It had occurred to him that perhaps the real significance and added safety of the newer anæsthetics was not being fully appreciated. An anæsthetist skilled in the use of ether would administer this drug without much worry for the surgical relief of exophthalmic goître, but to be set against this was the observation which he had frequently made, that in the hands of the same surgeon the immediate postoperative condition of these patients was many degrees better after nitrous oxide anæsthesia than after ether.

He was of opinion that nitrous oxide anæsthesia was essential for the most efficient handling of the victims of major accidents and public hospitals were failing in so far as they neglected to supply the facilities for its use. Mr. Newton had referred to the work of Dale, but the observation had been made clinically before Dale provided the experimental basis.

Dr. Lillies had referred to a certain formula which had been advocated as a means of deciding if a patient were "safe" for anæsthesia. This worked out very prettily, but he ventured to suggest that, given an opportunity for a good clinical examination, the errors in clinical estimates would not amount to more than 1%. He would be sorry to see clinical acumen lost in favour of what he felt disposed to describe as tricks with the sphygmomanometer.

In connexion with the provision of apparatus for nitrous oxide and oxygen anæsthesia in public hospitals the Branch should emphasize that the cost was not a consideration. Was it not anomalous that so much should be spent on "Insulin," provided in many instances for patients headed for death, while nitrous oxide anæsthesia was proscribed on the ground of expense?

DR. V. J. KINSELLA said that his remarks were offered in a spirit of inquiry. Previous speakers had expressed their belief that the gas anæsthetics were safer than ether and in support of that opinion Dr. Lillies had quoted the findings of some French investigators. Impressive examples of patients in desperate state who had survived operation under gas anæsthesia had been described. At the same time he could not help feeling with Dr. Davies that they must be cautious in accepting too readily the doctrine of the superior safety of these anæsthetics. Patients whose condition was desperate and apparently similar, had survived after ether anæsthesia.

He had been much impressed some years previously on finding that for several successive years not a single patient had died under anæsthesia at the Royal Prince Alfred Hospital in Sydney. Ether was used exclusively. The cases totalled some thousands and included those abdominal emergencies and street accidents which came to every large metropolitan hospital. Unfortunately he could not quote exact figures from memory.

Also authorities seemed to differ. He had recently read an article in the *British Journal of Anæsthesia* written by Mecker and Hines, of the Mayo Clinic, in which was reviewed the anæsthetic practice in that institution for the years 1922 and 1923. Ether had been used in over 14,000 operations and nitrous oxide-oxygen anæsthesia had been employed in something more than 2,000 operations. The conclusion reached was that while nitrous oxide was the safer anæsthetic in short operations, its mortality exceeded that of ether in operations lasting half an hour or more.

He could not help feeling, therefore, that whatever might be the other advantages of the gas anæsthetics, their superior safety for prolonged anæsthesia was as yet "non-proven."

DR. JAMES BOOTH advocated the retention of the preliminary injection of morphine in anæsthetic work. He had found by personal experience that the patient was much more comfortable and less affected by nausea and vomiting after the operation when morphine had been given.

The use of adrenalin in cardiac collapse had not been mentioned, but he was convinced of its value. He instanced the case of an apparently still-born child who was resuscitated by the intracardiac injection of adrenalin.

PROFESSOR F. C. WILKINSON said that he regarded nitrous oxide and oxygen as the most suitable anæsthetic for dental work, but at the same time much practice was required for its efficient administration. Further, having mastered the technique, the anæsthetist required to maintain himself in constant practice.

During the war he had acted as anæsthetist at a field hospital for a considerable time and his experience was identical with that of everyone else, that nitrous oxide-oxygen was the ideal anæsthetic in the presence of shock and sepsis. The manifestations of shock which so frequently appeared six or eight hours after ether anæsthesia, were eliminated by the use of nitrous oxide and oxygen. He would be interested to hear further discussion on the merits of nitrous oxide and oxygen for long operations.

DR. HOWDEN, in reply, said that he had tried most of the drugs recommended for preliminary medication, but he felt that the experience of the anæsthetists made more for the comfort of the patient than the preliminary injection.

DR. GREEN said that he had not seen any pulmonary complications after the intratracheal administration of ether to patients operated upon for exophthalmic goître. Tracheitis occasionally occurred, but this was not unusual in goître owing to the traction on the trachea made during the operation.

DR. W. S. NEWTON emphasized that the added safety claimed for nitrous oxide and oxygen did not so much concern the risk of the actual operation as the liability to shock supervening some hours afterwards. He had formerly experienced much trouble in obtaining satisfactory and pure nitrous oxide, but the gas was being imported from America in cylinders which fitted the machine.

DR. LILLIES said that he agreed that a preliminary injection of morphine made for the patient's greater comfort. His objection to it was based on the respiratory depression which it sometimes occasioned during the administration of the anæsthetic.

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THE ANNUAL GENERAL MEETING OF THE EYE, EAR, NOSE AND THROAT SECTION OF THE QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the B.M.A. Building, Adelaide Street, Brisbane, on December 1, 1926.

### Election of Officers.

The following office-bearers were elected for the ensuing year:

*President:* Dr. J. Lockhart Gibson.
*Vice-President:* Dr. W. N. Robertson.
*Committeeman:* Dr. Wallis Hoare.
*Honorary Secretary:* Dr. Arthur Murphy.

### Arrangements for Meetings.

It was agreed that the meeting of the Section should be held every quarter as heretofore. It was further resolved that two topics should be chosen for discussion at each meeting. Tobacco amblyopia and ethmoiditis were chosen as the subjects for the February meeting.

### Gunshot Wound of the Left Eye.

DR. J. LOCKHART GIBSON showed the enucleated eye of a returned soldier. He had first seen the patient on October 8, 1918, on his return from Egypt. On July 14, 1918, a bullet had entered between the right eye and the inner canthus, it had passed across the posterior part of the nasal cavity, across the back of the left orbit, destroying the left optic nerve and the posterior part of the left eyeball. It had passed out at the left temple.

On October 8, 1918, vision of the right eye had been confined to the perception of light and to tracing its direction, but photophobia had been present and it was necessary to keep the eye covered. Both eyes had been discoloured and bloodstained. The left globe had been shrunken and the eye totally blind. The scar in the right orbit had suggested that the external coats on the inner

aspect of the right eye had been either destroyed or indented. A coloboma of the iris had been present on the superior aspect, although no operation had been performed. An extensive rupture of the internal half of the chorioid had been present, the lens appeared to be clear, but the retina was detached in its greater part. Dr. Gibson had been unable to hold out any hope of improvement and the outlook was that perception of light would be lost. An offer of one thousand pounds had been made if vision could be restored, but Dr. Gibson had replied that he would be glad to pay that amount if he could perform such a miracle. The patient had not been satisfied and had sought advice elsewhere in Australia and, Dr. Gibson thought, in America. He had no doubt forced the hand of a much respected ophthalmologist to operate on him in London, apparently for the removal of a lens which had become opaque. The patient had returned to see Dr. Gibson on November 5, 1926, having seen no light for five years. The eye had been struck by some dry lucerne hay; it was inflamed and painful and apparently affected by panophthalmitis. Dr. Gibson had advised its removal. He drew attention to the dent on the internal aspect of the sclerotic and to the complete detachment of the thickened cup-like retina, except where the chorioid was so largely ruptured. At this site the retina was partly adherent to the sclerotic. The patient was leading an active and useful life.

### Glioma of the Optic Nerve.

Dr. Lockhart Gibson also showed a child of nearly eight years of age from whom he had removed both eyes for intraocular glioma of each optic nerve. At the time of operation the child's age had been seven months. The child was well and the sockets healthy. The removal of the eyes had therefore been worth while. The eyes had been shown at a previous meeting. Dr. E. O. Marks was endeavouring and apparently with some success to fit prostheses which up to that time had not been worn.

### Bronchoscopy.

A discussion took place on the bronchoscopic removal of foreign bodies from the lungs.

Dr. E. Culpin read the clinical notes of a child who was under his care and who had suffered from what seemed to be pneumonia in one lung. During a protracted convalescence the child had coughed up a peanut kernel. At this stage the child had been referred to him and the history revealed the fact that the child had inspired two kernels six weeks previously and that one had been recovered by the mother at the time. Dr. Culpin sought an expression of opinion as to the treatment in view of the improvement manifested since the expulsion of the second kernel. The members concurred in advising that bronchoscopy was undesirable.

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A meeting of the Queensland Branch of the British Medical Association was held at the B.M.A. Building, Adelaide Street, Brisbane, on February 18, 1927, Dr. H. V. Foxton, the President, in the chair.

### A Section of Surgery.

Dr. H. V. Foxton explained that the meeting had been called to consider the formation of a section of surgery in connexion with the Branch.

Dr. E. S. Jackson moved and Dr. Eustace Russell seconded:

> That it is desirable that a surgical section be formed in connexion with the Queensland Branch of the British Medical Association.

The following subcommittee was then appointed to investigate the matter and report later to the Council: Dr. G. P. Dixon, Dr. D. A. Cameron, Dr. L. M. McKillop, Dr. M. Geaney, Dr. A. V. Meehan, Dr. A. G. Anderson, Dr. V. McDowall, Dr. E. S. Jackson.

## NOMINATIONS AND ELECTIONS.

The undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

Godsall, John Rex., M.B., Ch.M., 1926 (Univ. Sydney), Pomeroy, Macleay Street, Potts Point.
Harrison, Charles William, M.R.C.S. (England), 1926, L.R.C.P. (London), 1926, F.R.C.S. (Edinburgh), 1926, The Sanitarium, Wahroonga.
Nicholl, Frederick Livingstone, M.B., Ch.M., 1925 (Univ. Sydney), Griffith Street, Manly.

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The undermentioned have been elected members of the Victorian Branch of the British Medical Association:

Moore, George Howard, M.B., B.S., 1926 (Univ. Melbourne), Camberwell.
Rothstadt, Margarita Charlotte, M.B., B.S., 1926 (Univ. Melbourne), Queen Victoria Hospital, Melbourne.

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Dr. F. A. Hope Michod has been appointed Chairman of Committees of the Queensland Branch of the British Medical Association.

## Medical Societies.

### THE NEWCASTLE HOSPITAL CLINICAL SOCIETY.

A meeting of the Newcastle Hospital Clinical Society was held at the Newcastle Hospital, New South Wales, on December 2, 1926, Dr. S. Gardiner, in the chair.

### Chlorosis.

Dr. Idris Morgan showed a girl, aged twenty years, the third child in a family of six, who was suffering from chlorosis.

No member of the family of either parent had suffered from anæmia or hæmophilia.

One brother had died of pneumonia at the age of twenty-three. Another brother had died in infancy; the mother was certain that this baby exhibited no symptoms suggestive of spontaneous hæmorrhage. A third brother had died at the age of one year. The mother's description aroused suspicion that the child had died from uncontrollable spontaneous hæmorrhage from the nose, mouth and bowel. She stated also that he "bled all over" and "big black blotches appeared under his skin." The doctor had told her that he would have been a bleeder.

Of the three children living one was "perfectly healthy," the second, a man of twenty-six years, bled from the nose frequently, but worked as a blacksmith. According to his mother he "is a big, strong, healthy chap, who is never ill." The third child was the patient.

When three years of age she had become ill, suffered from headaches, fainted frequently and was always "sickly." She had first been admitted to hospital at the age of four years. Her tonsils had been removed. The hæmorrhage which followed this operation, had persisted for such a long time that the child's life was in danger. The mother had been told that her daughter was a bleeder.

From this time on the child had always been ill; she was unable to go to school, suffered from frequent headaches and fainted after any unusual exertion, but there was no bleeding of any kind until the age of fourteen years. Dr. Idris Morgan said that this was an important point, the fainting, headaches and ill health persisted for ten years in the absence of hæmorrhage.

At fourteen years of age menstruation had commenced, the hæmorrhage was profuse at first and persisted continuously for seven weeks. The patient was prostrated and was obliged to remain in bed for two months. For the

last six years she had suffered from frequent hæmorrhages, generally from the nose.

Admission to hospital had frequently been found necessary. On two occasions the extraction of a tooth had caused bleeding for from ten to fourteen days. She had been treated by means of increasing doses of calcium lactate, injections of serum and defibrinated blood. The serum had given temporary relief from the spontaneous hæmorrhages.

At the time of demonstration she complained of menorrhagia, frequent epistaxis, dyspnœa on exertion, palpitations, frequent syncope, lassitude and giddiness.

Iron in increasing doses had not been prescribed. Blood had not appeared in the urine or fæces. There was no history of hæmoptysis or hæmatemesis.

The patient was one hundred and fifty-nine centimetres (five feet three and a half inches) high and weighed 51·75 kilograms (eight stone three pounds). There was a definite increase in the subcutaneous fat. The contours of the trunk and limbs were full and rounded. The skin of the whole body was waxy pale in colour. The lips exhibited the same pallor. The conjunctivæ were a definite blue colour. The area of cardiac dulness was slightly enlarged. A soft systolic bruit was heard at the apex and was not conducted to the axilla. There was slight œdema of the feet and ankles. The spleen was not enlarged. There was no enlargement of the lymphatic glands. Several large bruises were present, caused by very slight blows. The slightest knock or bump caused a subcutaneous extravasation of blood out of all proportion to the degree of violence of the blow. An examination of the other systems revealed no abnormality.

An examination of the blood (kindly carried out by Dr. Ethel Byrne) had yielded the following result:

| | |
|---|---|
| Erythrocytes, per cubic millimetre | 4,940,000 |
| Hæmoglobin value | 32% |
| Colour index | 0·33 |
| Leucocytes, per cubic millimetre | 5,600 |
| Polymorphonuclear cells | 63·2% |
| Basophile cells | 2% |
| Large lymphocytes | 22·5% |
| Large mononuclear cells | 0·5% |

Red cells manifested definite dimunition in hæmoglobin content with extreme degrees of poikilocytosis and anisocytosis.

Diffuse polychromatophilia was present. Two normoblasts were seen in films examined. There was a remarkable scarcity of blood platelets.

The coagulation time was eleven minutes (the method of Lee and White was used). When fragility was tested the blood cells had undergone hæmolysis in 0·4% saline solution.

Treatment had been commenced and iron pills in increasing doses were prescribed. No reaction had been obtained when the blood was subjected to the Wassermann test.

In discussing the differential diagnosis Dr. Morgan said that it became apparent from the narrative of the patient and her mother that practically all treatment had been based upon an endeavour to control hæmorrhage and prevent its recurrence.

Both the patient and her mother had been told on a number of occasions that the patient was a bleeder and on two occasions a definite diagnosis of hæmophilia had been made. In spite of all that might be written to the contrary, there were female bleeders. It might not be technically correct to say that they suffered from hæmophilia, but whatever name might be applied to the condition, the treatment presented the same problem.

The appearance of the patient, the undiminished subcutaneous fat, the slight enlargement of the heart, the absence of pyrexia, the fainting turns and the blood picture supported the diagnosis of chlorosis.

Menorrhagia and epistaxis were not very commonly met with in chlorosis, but did occur in severe cases. Normoblasts might also be found. The blood picture was one which might be expected in a severe case of chlorosis or in a person who was the subject of frequently repeated losses of blood. It had been pointed out that frequently repeated losses of blood produced an anæmia of the chlorotic type.

The commencement of the symptoms at the age of four years was evidence against the diagnosis of chlorosis. On the other hand the persistence of the symptoms without hæmorrhage from the age of four to fourteen years was very suggestive of a primary anæmia.

The absence of any history of "bleeders" in the families of the parents was counterbalanced by the condition which caused a brother's death at the age of twelve months.

A history of melæna, hæmatemesis, hæmaturia and joint involvement would be expected in a case of hæmophilia presenting symptoms so severe and persistent as those of the patient. Spontaneous hæmorrhage had always been from the nose. Thursfield remarked that epistaxis was uncommon in chlorosis, but hæmorrhage from other sources did not occur.

The almost complete absence of blood platelets was suggestive of a primary anæmia.

The blood count and physical signs strongly suggested chlorosis. The history suggested hæmophilia or primary anæmia. The effect of gradually increasing doses of iron upon the symptoms and blood picture was being used as a guide to the correct solution. Chlorosis responded well to such treatment.

Blood examination in November, 1926, had yielded the following results:

| | |
|---|---|
| Erythrocytes, per cubic millimetre | 4,000,000 |
| Hæmoglobin value | 45% |
| Colour index | 0·56 |

No nucleated forms were seen. Poikilocytes and anisocytes were present. Neutrophile cells numbered 71% and lymphocytes 28%. The eosinophile cells were 0·5%.

Blood examination on December 31, 1926, had yielded the following results:

| | |
|---|---|
| Erythrocytes, per cubic millimetre | 4,820,000 |
| Hæmoglobin value | 60% |
| Colour index | 0·6 |
| Leucocytes, per cubic millimetre | 7,200 |
| Polymorphonuclear cells | 79% |
| Eosinophile cells | 2% |
| Basophile cells | 1% |
| Large lymphocytes | 2% |
| Small lymphocytes | 16% |

The red cells had manifested diminution in hæmoglobin content, with variation in size and shape. No polychromatophilia was present and no nucleated red cells were seen.

### Postoperative Hemianopsia.

DR. F. W. D. COLLIER presented a patient who was suffering from hemianopsia following an operation for epithelioma of the lower lip. The patient, a wharf labourer, aged fifty-two years, had been admitted to Newcastle Hospital on October 13, 1926, complaining of a sore on the left lower lip which had been present for four months. He stated that about four months before his admission a cigarette paper had stuck to his lip and when he pulled it away, "a piece of skin came away with it." Soon afterwards a sore had appeared on the lip in the site of this lesion. It had increased in size. A small swelling had appeared on each side of the lower jaw fourteen days prior to the day of the meeting. An examination revealed two small lesions on the right side of the lower lip both covered with a yellow crust. The medial lesion had been indurated. A small, hard gland could be palpated in each submaxillary triangle. On October 14, 1926, a complete removal of the cervical glands on the right side and the glands of the left digastric triangle had been carried out. On October 28, 1926, a V-shaped portion of the lower lip including the site of the lesion had been resected. Three days after the first operation the patient had noticed that he was unable to see anyone standing on the left side of his bed. No evidence of arteriosclerosis or other disease of the vascular system had been detected. The systolic blood pressure was 140 millimetres of mercury and the diastolic pressure 100 millimetres.

Dr. R. Candlish had examined the eyes and discovered the presence of a left-sided homonomous hemianopsia. The blind areas had been extended to fixation point. The fundi were normal in appearance and no abnormalities of function could be discovered in the cranial nerves. The hemianopsia had persisted to the date of the meeting. The lesion was probably a thrombosis and was situated between the optic chiasma and the occipital lobe or in the optic radiation on the right side. No general disturbance accompanied the hemianopsia. A lumbar puncture had not been carried out. It was suggested that a Wassermann test should be performed upon the cerebro-spinal fluid. The discussion centred round the question of the pathological process causing the lesion.

### Case for Diagnosis.

DR. A. C. ARNOLD showed a male patient whose illness had commenced three weeks prior to the date of the meeting with a sore throat and headache. The patient maintained that he had not felt ill in spite of the fact that for three weeks his temperature had been elevated from 2° to 3° F. above normal and was still elevated.

The temperature had been of the "swinging type." Typhoid fever had been excluded. A blood examination on admission revealed a leucocytosis (26,000) with a relative increase of polymorphonuclear cells.

Three weeks later the white cells had numbered 16,000 per cubic millimetre. Secondary anæmia had not been present. About ten days previously the lymphatic glands of the axilla, groins and triangles of the neck had commenced to enlarge. A provisional diagnosis of Hodgkin's disease had been made and arsenic had been exhibited. The temperature had fallen two days prior to the day of the meeting. The tonsil of the left side was still enlarged and looked very unhealthy. During the discussion it was suggested that the leucocytosis, the elevated temperature and the rapidity of the enlargement of the lymphatic glands were more indicative of toxic adenitis than of Hodgkin's disease.[1]

### Pseudo Tabes Dorsalis.

DR. J. BOSTOCK presented a man, aged forty-three years, who complained of "giving way at the knees and loss of power in the legs for twelve months." The patient also complained of dyspepsia, attacks of diarrhœa, pain in the upper part of the abdomen and præcordial and fairly severe pains in the limbs, back and neck.

This patient had a definitely low intelligence quotient. Three other members of the family suffered from a similar condition. The patient stated that some of his cousins exhibited the same symptom, but not in such a marked degree.

No consanguinity existed between the parents of this family. On examination the following signs and symptoms were noted: Complete absence of knee jerks, sluggish reaction of the pupils to light, a gait suggestive of tabes but not actually typical, slow, sluggish speech and dull mentality.

Radiological examination revealed a condition of general visceroptosis. The Wassermann test had been applied to the blood and cerebro-spinal fluid and no response had been obtained. An examination of the blood had revealed a slight secondary anæmia. A diagnosis of *pseudo tabes dorsalis* had been made.

During the discussion it was suggested that the condition might have been the result of congenital cerebral deficiency.

### Pathological Specimens.

DR. E. TRENERRY exhibited a number of pathological specimens. The first was portion of the lower bowel of a male patient, aged thirty-six years, who had been admitted suffering from acute bronchitis. During convalescence a sudden and severe hæmorrhage from the bowel had occurred and had caused death. The bowel was pitted with numerous chronic ulcers typical of bacillary dysentery.

The heart and aorta of a male patient, aged forty years, exhibited a condition of advanced atheroma of the wall of the aorta and aortic valves. A Wassermann test had been applied to this patient's blood and had given a triple positive response.

The third specimen consisted of a double ureter and double kidney. The patient from whom these specimens had been removed, had been admitted to hospital in a moribund condition due to cerebral hæmorrhage and had lived only a few hours. At the *post mortem* examination the left kidney and two tubular structures running from the kidneys to the bladder had been removed, together with a portion of the bladder wall. The pelvis of the kidney had been much enlarged and cystic in appearance. One of the tubular structures mentioned above was undoubtedly a ureter. The other structure resembled a ureter and a probe could be passed from the pelvis into the lumen of this structure. A large bristle passed easily from the lumen at the other end into the bladder.

During the discussion it was pointed out that a definite constriction was present towards the upper portion of the enlarged kidney.

A further examination showed that the specimen contained two kidneys and two ureters with the common pelvis. The ureters opened separately from the pelvis, but entered the bladder by a common orifice.

## THE MELBOURNE HOSPITAL CLINICAL SOCIETY.

A MEETING OF THE MELBOURNE HOSPITAL CLINICAL SOCIETY was held at the Melbourne Hospital on March 25, 1927, MR. ALAN NEWTON, the President, in the chair.

### ? Supra-Pituitary Neoplasm ? Idiopathic Narcolepsy.

DR. H. F. MAUDSLEY showed a male patient, aged nineteen years. His sister had died at the age of fourteen years of juvenile general paralysis of the insane. He had been in good health until three years previously when he had dived into shallow water. He had felt no ill effects at the time, but three days later began to suffer from attacks of sleep (narcolepsy), which he was quite unable to control. Lately the attacks had become much more frequent, they had at times been preceded by an olfactory aura and during them he had occasionally fallen down.

Under the stress of any emotional stimulus his muscles became quite flaccid and he fell down, although he did not lose consciousness. Owing to these attacks of catalepsy he was quite unable to attend a cinema. During the past two years he had gained nineteen kilograms (three stone) in weight. All sexual feeling had disappeared. His eyesight was poor. His intelligence was good. He did not complain of any other symptoms.

On January 20, 1927, Dr. Miller had reported slight pallor of the left optic disc and some contraction of both visual fields. A month later there was definite contraction of both fields and temporal pallor in both discs. There were no other neurological signs. The blood had failed to react to the Wassermann test. X ray examination of the skull revealed nothing abnormal. The basal metabolic rate was normal. There was no polyuria, but the sugar tolerance was slightly raised. Adie had collected twenty cases of idiopathic narcolepsy, including six which he had personally observed. In all of these the patients were subject to uncontrolled attacks of sleep and cataleptic phenomena, exactly similar to those seen in the patient presented. He considered that catalepsy and narcolepsy were ætiologically related. Redlick had also reported cases characterized by disorder of the ductless glands. Dercum, Henenberg and Streiffer had demonstrated symptoms indicating pituitary disorder in some of their patients. Adie, using experiments similar to those of Pawlow in his researches on conditioned reflexes, had been able to induce sleep in dogs by gradually increasing the interval between the stimulus and the expected food. He concluded that the continued cortical stimulation had spread to the supposed sleep centres in the "tween-brain." In man such

[1] *Later*: The enlarged glands gradually assumed their normal size and the symptoms disappeared.

spread might take place in pathological conditions from any cortical point or pathological changes, such as encephalitis, pituitary or suprapituitary tumour in the region of the "tween-brain," might give rise to sleep attacks. Dr. Maudsley considered that in view of the well defined attacks of narcolepsy and catalepsy the abnormal increase in weight and the changes in the optic discs and visual fields, a diagnosis of suprapituitary tumour was probably correct, and he asked for an expression of opinion as to whether operative interference was justified.

Dr. R. Ellery who had previously seen the patient with Dr. Maudsley, said that the patient had also had some headache above the left eye. Lately cataleptic attacks had occurred without any preceding emotional stimulus. He had at first regarded the condition as one of idiopathic narcolepsy, but in view of the subsequent developments he considered the diagnosis of slowly growing pituitary neoplasm to be more probable.

Mr. Alan Newton, in discussing the method of surgical approach, said that for pituitary tumours the transnasal route was preferable. The risk of sepsis was not so great as usually supposed. For suprasellar tumours, however, he would advise the frontal route, care being taken to lift up the frontal lobes and *dura mater* together, the latter being incised far back.

Dr. G. Pennington thought that in spite of the absence of response to the Wassermann test intensive antisyphilitic treatment should be undertaken, seeing that the patient's sister had died of juvenile general paralysis of the insane. He instanced a case of *encephalitis lethargica* in which attacks of narcolepsy had ceased after the development and drainage of an ischio-rectal abscess.

Mr. C. H. Osborn said that Dott had recorded several cases similar to the one presented. In all of these a suprasellar tumour had been found and the symptoms had been due, not to pituitary disease, but to pressure on the "tween-brain." He thought that a suprasellar tumour was probably present.

Dr. Maudsley, in reply, thanked members for their suggestions and instanced a patient with narcolepsy in whom undoubted evidences of *encephalitis lethargica* had subsequently developed.

### Aneurysm of the Descending Aorta.

Dr. S. O. Cowen showed a male patient, aged sixty-two years. In May, 1926, he had had a hydatid cyst removed from the adductor region of the thigh. In August, 1926, he had begun to complain of pain in the chest and dyspnœa on exertion. On examination there were systolic and diastolic bruits over the aortic area. There was one finger's breadth of dulness to the right of the sternum in the second intercostal space. In the left interscapular region there was an area of dulness over which the heart sounds were audible. X ray examination revealed a rounded shadow in the mediastinum, about ten centimetres (four inches) in diameter and not pulsatile, which was reported as being probably a hydatid cyst. The Casoni and complement fixation tests for hydatid both yielded positive results. The blood reacted strongly to the Wassermann test. Physical examination revealed no other abnormality. The patient had been operated on by Mr. Dew, who found a pulsating aneurysm with a very thick wall, arising from the descending aorta.

Dr. Cowen pointed out that all patients with syphilis should be carefully examined for signs of aortitis which, if found, should lead to a suspicion of aneurysm and a full X ray examination. About 50% of patients with aortitis showed some neurological signs of syphilis. About 30% of aneurysms were reported by the radiologist as being non-pulsatile. This was usually attributed to the presence of a thick sac wall, but in many cases it was due to surrounding mediastinitis which after antisyphilitic treatment might resolve to such an extent that pulsation, previously not present, might then become visible. This had been so in the patient presented. In all cases of cardiac syphilis he began treatment with a preliminary course of mercury and iodide of potassium. He then gave "Arsenobenzol," commencing with a dose of 0·1 gramme.

Mr. H. R. Dew said that the hydatid of the thigh was an old standing lesion. If the lung had been infested at the same time, he would have expected that the cyst would have been either very large or calcified. As the X ray examination had revealed a comparatively small shadow with no evidence of calcification he thought that hydatid disease should have been considered as being improbable.

Dr. H. Turnbull said that the only way to make an early diagnosis of aneurysm was to suspect it in all patients with syphilitic aortitis. In his experience more than 50% of aneurysms were associated with signs of aortic regurgitation. The aortic valves, however, might be quite free from signs of disease and the aneurysm arose in the aorta beyond. He thought that antisyphilitic treatment was useful, particularly in early cases, and quoted one in which the aneurysm had developed seven months after the original infection. He was very sceptical about the value of treatment in late cases, for in these it was very difficult to assess the results. The natural course of the disease varied considerably and he instanced one patient who had had no treatment and was still well fourteen years after hæmoptysis.

### Foreign Body in the Lung.

Dr. F. B. Lawton showed a male patient, aged fifty-two years. He had been presented at the October meeting of the Society in 1926 as suffering from bronchiectasis. At that time arrangements had been made for X ray examinations after the injection of "Lipiodol" and for bronchoscopic examination. Three weeks before the appearance of symptoms which led to his consulting a doctor, he had thought that he had "swallowed" a bone. X ray examination after the injection of "Lipiodol" had confirmed the diagnosis of bronchiectasis. Mr. Scantlebury had made two bronchoscopic examinations. On account of the large amount of pus, the result of the first examination had been indefinite, but at the second a rabbit's vertebra had been found and removed. Since then the patient had slowly improved and had been able to do his work as a painter for the last two months. He had put on four kilograms (nine pounds) in weight. He had been treated by postural drainage and had been given a mixture containing potassium iodide and creosote. The amount of sputum had become much less, but had increased considerably after an injection of "Lipiodol," given one week previously. Dr. Lawton asked for an expression of opinion as to whether any other form of treatment should be employed. He had considered inducing artificial pneumothorax, but felt doubtful about its success, as he thought that extensive pleural adhesions would be met with and he doubted if it would cause collapse of the dilated bronchi. He thought, however, that it might be worth trying.

Mr. C. Scantlebury said that after the pharynx and larynx were swabbed with local anæsthetic, the curved needle of a syringe had been passed through the vocal cords and twenty cubic centimetres of "Lipiodol" injected. He thought that the apparent increase in the bronchiectasis shown in the second X ray plate was probably due to the entry of more "Lipiodol" and not to any increase in the bronchial dilatation. At the first bronchoscopic examination a good deal of pus had been swabbed out, and the cavity had then been filled with argyrol. At the second examination the vertebra had presented with the spinous process uppermost and was removed.

Mr. Harold Dew did not think that any surgical interference was called for.

### Generalized Glandular Enlargement.

Dr. Douglas Thomas showed a male patient, aged sixty-two years. He had been operated on some years previously for gall stones. On January 1, 1927, he had noticed some soreness and swelling in the left axilla. Two or three days later a swelling appeared in the right axilla and this was soon followed by swellings in the neck and groin. On examination there were large glands in each axilla, the largest being about the size of a hen's egg. They were fairly firm, discrete, freely movable and tender only on deep pressure. There were smaller glands in both sides of the neck and groin. X ray examination of the chest revealed nothing abnormal. The blood had failed

to react to the Wassermann test. On January 8 an examination of the blood had revealed a hæmoglobin value of 80%. The leucocytes had numbered 8,000 per cubic millimetre and of these the polymorphoneutrophile cells had numbered 24% and the lymphocytes 76%.

The stained film contained no abnormal cells. A further examination of the blood made on March 1 had yielded the same findings as before, except that the lymphocytes were reduced to 28%. Microscopical examination of an excised gland revealed changes which the pathologist regarded as being suggestive of Hodgkin's disease. In the previous few weeks the glands had rapidly decreased to about one half their former size. Dr. Thomas considered that the diagnosis was either Hodgkin's disease or glandular enlargement from some undiscovered infection. Tuberculous and chronic infective glands might bear close resemblance to Hodgkin's disease with which they were sometimes confused. He believed that there were conditions simulating Hodgkin's disease and running a benign course in which X ray treatment gave good results. He asked for an expression of opinion as to the diagnosis and the advisability of giving X ray treatment.

Dr. S. O. Cowen said that if he had seen the patient in the earlier stages of the disease, when the percentage of lymphocytes was high and the leucocyte count was low, he would have favoured a diagnosis of lymphatic leuchæmia. He had seen two patients suffering from this disease whose blood had contained not more than 11,000 leucocytes per cubic millimetre of blood. The subsequent developments had made this diagnosis untenable. He thought that the enlarged glands were due to an atypical lymphadenitis with a blood picture allied to infective mononucleosis, although there was present none of the classical features of this disease. He considered that the possibility of both syphilis and tuberculosis should still be borne in mind.

Dr. Keith Fairley said that in his opinion the glandular enlargement was due to an infection, although in the earlier stages he would have seriously considered the diagnosis of lymphatic leuchæmia. He thought that tuberculosis could be excluded and that in view of the absence of reaction to the Wassermann test syphilis was most unlikely. He had seen four children suffering from infective mononucleosis, in all of whom a diagnosis of lymphatic leuchæmia had been originally made.

Dr. L. E. Hurley said that only about 25% of cases with a clinical picture of Hodgkin's disease were characterized by the typical histological structure in the glands. He had seen two patients diagnosed as suffering from Hodgkin's disease after the microscopical examination of an excised gland, in whom the enlargement of the glands had subsided, and who still enjoyed good health four years later. In the diagnosis of lymphatic leuchæmia from lymphocytosis the amount of change in the red cells and the internal structure of the lymphocytes were important factors. In lymphatic leuchæmia the lymphocytes (lymphoblasts) had primitive nuclei with delicate chromatin network and several nucleoli.

Dr. Hume Turnbull said that he thought the glands would subside. He considered that 74% of lymphocytes was hardly high enough for a diagnosis of lymphatic leuchæmia.

Dr. Richard Stawell said that in elderly people the cause of general glandular enlargement was very difficult to diagnose. He had seen at least twelve in whom the glands had rapidly appeared as in this case and had then subsided. When the blood picture was not characteristic, he had usually regarded the condition as Hodgkin's disease, the exact nature of which was not yet clear, but which was probably an infective lymphadenitis. He thought that cases of general glandular enlargement could be divided into two groups according to the age of the patient. A good prognosis could be given for patients over fifty years of age, provided there were no Pel Ebstein paroxysms. In patients under thirty the prognosis was not so good and they were difficult to control. In the patient presented he thought that the prognosis was good and that X ray treatment to the glands might hasten their subsidence.

# Correspondence.

## THE RED-BACKED SPIDER BITE.

Sir: Dr. Miles has recorded some of his experiences in the treatment of cases poisoned by the red-backed spider and asks for an opinion on future treatment. I think that one can indicate a reasonable line to follow once some idea of the nature of the venom has been disclosed.

Many spiders have an evil reputation which they do not deserve and the most maligned is the *Tarantula*. The evil effects that were supposed to arise from its bite are but a myth; the symptoms that followed the bite of the *Tarantula* back in the dark ages were due to a kind of epidemic hysteria (tarantism) and were not due to any venom injected. The subjects having been bitten imagined that their only hope lay in dancing until they became exhausted. The rapid dance was called the Tarantella, and so the notion arose that this mad kind of dancing was produced by the action of the venom of the *Tarantula*.

Kobert in 1901 was, however, able to show that tarantulas are harmless and that real poison was found in only two genera of spiders, namely *Lathrodectes* and *Epididæ*, and it is to the latter genus that the garden spider (*Epeira diademeta*) belongs.

The *Lathrodectes* are distributed over the whole world, being found in Italy, South Russia and South America and our red-backed spider (named after Van Hasselt, "*Lathrodectus hasselti*") belongs to this genus. It has a round, black body with a bright scarlet stripe running from front to rear on the dorsal surface of the abdomen. As far back as 1765 Valmont de Bombare gave a description of the poisonous properties of the Malmignatte, a species of *Lathrodectes* found in Italy, and since then this spider has frequently been studied. But our chief knowledge of the effects of spider venom we owe to the labours of Kobert who has made many experiments with aqueous extracts of the Crimean *Karakurtes*, also belonging to the *Lathrodectes*. These extracts were found to be very poisonous and their intravenous injection produced the same symptoms as those observed in men bitten by these spiders. The chief symptoms were dyspnœa, convulsions and paralysis of the respiratory system and heart, speedily ending in death. I might here remark that tick venom will produce all these symptoms, as it has a selective action on the vagus centre. It is curious to note that new-born spiders were more venomous than full-grown ones: this does not hold good for ticks.

Kobert's further researches were made on the common garden spider (*Epeira diademeta*) and he found in this spider a very analogous poison to that found in the *Lathrodectes*. He also discovered that the poison of this spider possesses hæmolytic power and this point was further investigated by Hans Sachs (1902) who found that the toxin in the venom, called by him arachnolysin, has a very rapid and intense solvent action upon the blood corpuscles.

In working out the way that the venom of the dog tick affects animals, I have come to the conclusion that snakes, spiders, ticks and scorpions have in their venoms toxins which act in the same way when introduced into other animals. Just as various species of snakes have different combinations of toxins in their venom—no one snake having every kind of toxin found among all venomous snakes—so it is with spiders, ticks and scorpions, all of which are closely related, but they are grouped under different orders of the class *Arachnida*. Some of these have two or more toxins in their venoms; others have two but they are different toxins. I may perhaps make this point clearer by saying that snake venom is a complex substance containing neurotoxins, anticoagulin, hæmorrhagin, thrombase and digestive ferments, the latter being classified into groups as cytolysins, proteolytic ferments and kinases.

Now when we come to study tick venom we find that its chief constituent is a neurotoxin having a specific action on the vagus centre and in addition it contains anticoagulin. Spider poison, while it contains a neurotoxin which has an irritant action upon the heart and also on the central nervous system, this leads to paralysis;

in addition to this there is a toxin called arachnolysin, which has an intense action on the blood corpuscles and this resembles the action of the hæmolysin of cobra venom.

Kyes was able to isolate from scorpion venom a lecithide possessing an immediate blood solvent action and exactly analogous to that obtained from cobra venom.

Calmette came to the conclusion that scorpion venom differs no more from snake venom than do these from each other and also like them it contains the neurotoxin of the snakes.

Wilson found that scorpion poisons are probably members of the diphtheric group of poisons and must be closely allied to snake venoms. He also found that the poison affected the muscular system by acting on the peripheral nerve endings, not on the central nervous system and I have evidence that the paralysis produced in the dog's legs by tick venom is partially if not wholly due to a peripheral neuritis.

We are now in a position to attack the question of how to treat spider bite. I think I have in the limited space allowed me produced sufficient evidence to show that the tick, the spider and the scorpion all have in their venoms toxins which act like the toxins found in snake poison and I strongly urge the view that there will one day be found similar toxins in the venoms of all these four groups of animals. That being so, it seems to me that the most reasonable and logical way of attacking spider bite is to use those remedies that have been found efficacious in the local treatment of snake bite in India. Experience has shown that if the spot where the venom is injected by the snake be treated without delay that the venom can be made almost harmless. The one essential point is to act quickly. If delay occurs the venom is absorbed and acts on the central nervous system or the blood. Of all the remedies that have been tried—among them being permanganate of potash, chromic acid, hypochloride of lime, and ammonia—chloride of gold has proved the most successful. I would, therefore, suggest in treating spider bite that ten cubic centimetres of a 1% solution of gold chloride be injected, and by this means we may hope to reduce to a minimum the amount of venom that will be absorbed.

Dr. Miles tells us that there is an intense burning pain at the site of the lesion—this I might mention is characteristic of viper snake poison—and that the general toxic symptoms appear after a period of two or three hours; consequently an effort must be made to inject the chloride of gold during the first hour. The baby that died recently is said to have expired ninety minutes after being bitten. It may be that the spider injected the venom into a blood vessel; when the mode of injection is carried out with some of the other venoms death may take place in a few minutes.

Yours, etc.,

STEWART MCKAY.

Sydney,
March 6, 1927.

## HYPNOTIC SUGGESTION.

SIR: I am at a loss to understand what Dr. R. A. Parker means by the "secret hypnotist" and the "secret power." These phrases smack somewhat of the denunciation against hypnotism which used to be hurled by Mrs. Mary Eddy, but they have really no significance.

Again Dr. Parker quotes someone as saying: "Many persons have become mentally deranged and physically worse for improper treatment of their subconscious minds."

But Dr. Parker does not inform us what the "improper treatment" consisted of. Perhaps it was not hypnotic suggestion at all, but psychoanalysis which is anathema to some psychologists. I can believe that these stage exhibitions of persons in the hypnotic state may lead to mental disturbance if long continued, but that has nothing to do with the scientific use of the method. As for murder and other crimes committed under hypnotic influence, these are found mostly in the pages of Phillip Oppenheim and Gaston Leroux, though in France the plea of undue influence has been put forward in the courts without much success.

As regards permanence of cures, I can only say that some of my drunkards and insomniacs of twenty years ago and more are still well, but of course, as I took care to affirm before, they may be only "*post hocs*." After using this procedure off and on for more than thirty-five years, I cannot remember any evil effects, though in one case the result was so curious that it is worth relating.

It was that of a highly cultured woman, Scotch by birth, but educated in France and England. She was equally conversant with both languages. I hypnotized her for facial neuralgia and on rousing her from the hypnotic state, found to my amazement and dismay that she was amnesic as far as English was concerned. She could, however, chatter freely in French. Her English only came back slowly and it was about ten days before she was mistress of it again. A second trial, although combined with energetic suggestions against this lapse of memory or whatever it was, produced the same result, only this time the English was recovered in two or three days.

Can the psychologists explain this case?

Yours, etc.,

RICHARD ARTHUR

Macquarie Street, Sydney,
March 4, 1927.

## THE ANATOMY OF THE FEMALE PELVIS.

SIR: The anatomy of the female pelvis is of such vital importance to the welfare of the future mothers of the race and to the coming generations of obstetricians and gynæcologists, upon whom such welfare will largely depend, that I feel constrained to make some further comment on the portion of Dr. Maguire's thesis which appears to need reconstruction.

And as a preliminary, it is not perhaps a mere truism only to say that anatomists of today should be as absolutely accurate as the present state of knowledge allows and that obsolete or discredited theories should be rigorously excluded from their teaching.

Dr. Maguire rejects the fact enunciated by the late Arthur Nyulasy in 1914, namely that "the cardinal ligaments are the main supports of the uterus," a fact absolutely confirmed five years later by the well known author of the genito-urinary section of Cunningham's anatomy, Professor A. F. Dixon, and by other authorities.

In place of this fact Dr. Maguire substitutes the antiquated, but now exploded theory, that "the uterus is supported by the sheaths of its blood vessels." This theory was first put forward by the anatomists, Cameron and Derry, independently in the *Journal of Anatomy and Physiology*, London, 1907.

The views of these anatomists were brought before the Obstetrical Section of the Royal Society of Medicine in December, 1907, by the late Professor Fothergill and his most important statement, based on their teaching, was that the support of the uterus "is accompanied by the fibrous sheaths of the blood vessels and accompanying structures which supply the pelvic viscera."

In closing the discussion, however, the President of the Section, Dr. Herbert Spencer, one of the ablest gynæcologists in the United Kingdom, said that "anyone who had performed a total abdominal hysterectomy . . . would find it difficult to accept the statement that it was the sheaths of the vessels that kept the uterus in place." Probably the same difficulty will present itself to most of your readers.

And Professor A. F. Dixon is still more emphatic in his disapproval of the idea. Yet this is the theory now revived by Dr. Maguire. He says the neurovascular bundles of the pelvic connective tissue are the most important of the direct supports of the uterus.

There is another point I should like to make quite clear, once for all. The cardinal ligaments are frequently referred to as "Mackenrodt's," but this author's description is quite erroneous, for he describes them as "bands of firm fibrous tissue, which form specialized parts of the pelvic fascia; arising in the neighbourhood of the ischial spines,

they pass through the pelvic cellular tissue and are inserted into the sides of the cervix and vaginal vault."

Arthur Nyulasy's original dissections, as described in THE MEDICAL JOURNAL OF AUSTRALIA in 1914 prove that these ligaments are not mere bands of firm fibrous tissue, they are not specialized parts of the pelvic fascia, they do not arise in the neighbourhood of the ischial spines and they are not inserted into the sides of the cervix and vaginal vault. On the contrary, they arise from the uterus and pass outward to be inserted into the pelvic fascia on the lateral wall of the pelvis and they are packed (as before stated) with bundles of smooth involuntary muscular fibres, whose presence Mackenrodt quite overlooked.

Five years after the publication of Arthur Nyulasy's papers his views on the structure and functions of the cardinal ligaments were absolutely confirmed by Professors Hastings Tweedie and A. F. Dixon at a meeting of the Royal Academy of Medicine in Ireland.

It would thus appear that the name of "Mackenrodt" should be dropped altogether in connexion with these ligaments and as Victor Bonney remarked in 1923, "it is time there was a consensus of opinion as to the nomenclature of such important structures."

They have also been called the cardinal ligaments of Kocks, but this writer referred to them merely as the "bases of the broad ligaments," without giving a single detail of their anatomy. Here are his words: "The bases of the *ligamenta lata* play such an important part in the attachment of the uterus, that they may with more justice be called special ligaments than the *ligamenta rotunda*. They form the axis of the uterus and might on account of their importance be called *ligamenta cardinalia*." In other words, he supplied the name, but Arthur Nyulasy described the anatomy, of the cardinal ligaments.

It may be added that in 1914, at the invitation and in the presence of Professor A. Watson (formerly Professor of Anatomy and of Operative and Experimental Surgery in the Adelaide University), Arthur Nyulasy made a dissection in the Medical School to fully expose the cardinal ligaments. So impressed was the professor with the originality and accuracy of the demonstration that he more than once afterwards referred to the ligaments as "Nyulasy's ligaments"—a name by which I venture to hope and believe they will in future be generally recognized.

Yours, etc.,

FRANK A. NYULASY.

Melbourne,
March 21, 1927.

## "BISMOL" IN SYPHILIS.

SIR: In your issue of the twelfth instant, page 384, Dr. Southby, replying to a question by Dr. Forsyth, said "he was sorry to state that 'Bismol' was not being manufactured." Will you kindly permit us to explain that since we introduced "Bismol," its reception by the medical faculty has been so encouraging, that production has been continuous.

Stocks of freshly prepared "Bismol" are always available, as well as professional samples for practitioners who are interested.

Yours, etc.,

FELTON, GRIMWADE & CO., PTY., LTD.,
R. E. WOOD, General Manager.

342-346, Little Flinders Street,
Melbourne,
March 22, 1927.

## CONSANGUINEOUS MARRIAGES.

SIR: Will you kindly allow me to correct a statement attributed to me. On page 488 of the issue of March 26 I am represented as saying that degeneracy had been found in the Pharaohs of the Eighteenth Dynasty.

The late Sir Armand Ruffer pointed out that the marriage of brother and sister or half-brothers and half-sisters was the rule in ancient Egypt so far as the ruling class was concerned. It may have been more general The custom prevailed amongst the Ptolemies. It was brought about by political conditions and the inheritance of the throne, at all events technically, through the female. Yet amongst the great rulers of the Eighteenth Dynasty and also the Ptolemaic sovereigns there is no evidence of any degeneration. Thotmes III, the greatest soldier and statesman Egypt produced, was a product of a consanguineous marriage.

The conclusion reached was that, given sound stock, consanguineous marriages are not liable to produce degenerates.

Yours, etc.,

JAMES W. BARRETT.

105, Collins Street, Melbourne,
March 28, 1927.

## ANDREW HONMAN.

SIR: In the December 4 number of your journal I learned just now of the death of Doctor Honman.

During the year 1921 I had the pleasure and profit of serving under the old Colonel in New Guinea and learned to have the deepest respect for his ability and a great affection for him personally. He commanded loyalty from those under him by his loyalty to them. An attack on a subordinate or his plans would evoke a fiercer defence from the Colonel than one on his own. His friends were trusted implicitly by him and they rarely let him down. His reorganization of the medical system in New Guinea always seemed to me especially admirable.

Colonel Honman went to New Guinea at the age of sixty-three and started out to survey his territory as soon as he could, as a good strategist should. I made many of these trips with him and old as he was and young and fit as I was and accustomed to similar work in Papua, there was many a time when I was exhausted and the old Colonel seemed as willing as ever to go on. He may have been an invalid when young, but he was tough timber at sixty-three.

His work as a surgeon I saw little of, but in organization and administration he was an unusual man. He was brimful of ideas and energetic in mind and body; a hard worker, a loyal friend and a delightful companion. Few men die leaving the rounded life of achievement that Andrew Honman left.

Yours, etc.,

S. M. LAMBERT.

Suva, Fiji,
February 28, 1927.

# Naval and Military.

## APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Numbers 19, 21, 22, 30, 33 and 34 of February 24, March 3, 10, 24, 31 and April 7, 1927.

### PERMANENT NAVAL FORCES OF THE COMMONWEALTH (SEA-GOING FORCES).

*Confirmation in Rank.*—Surgeon Lieutenant (on probation) Robert Nihill, M.B., Ch.M., is confirmed in the rank of Surgeon Lieutenant, with seniority in rank of 12th March, 1926.

*Confirmation in Rank.*—Surgeon Lieutenant (on probation) Francis Joseph Matthews, M.B., B.S., is confirmed in the rank of Surgeon Lieutenant, with seniority in rank of 12th March, 1926.

### ROYAL AUSTRALIAN AIR FORCE.

**Permanent Air Force (Medical Branch).**

*Appointment.*—Short Service Commission of four years as Flight Lieutenant (on probation), 1st February, 1927. Walter Duff Counsell.

*Resignation Accepted.*—Flight Lieutenant E. H. W. Deane, 31st January, 1927.—(Ex. Min. No. 47.)

AUSTRALIAN MILITARY FORCES.

**Australian Army Medical Corps (Permanent).**

Colonel (temporary Major-General) G. W. Barber, C.B., C.M.G., D.S.O., V.D., to be Major-General.—(Ex. Min. No. 76.)

**First Military District.**

*Eleventh Mixed Brigade, Staff.*

Lieutenant-Colonel G. W. Macartney, D.S.O., Australian Army Medical Corps, is appointed Assistant Director of Medical Services, 5th Divisional Head-Quarters, and is granted the temporary rank of Colonel whilst holding such appointment, 1st February, 1927.

*Australian Army Medical Corps.*

Honorary Captain M. L. D. McKeon is appointed from the Australian Army Medical Corps Reserve and to be Captain (provisionally), 4th February, 1927.

Captain T. K. R. Allen is transferred from the Australian Army Medical Corps, 3rd Military District, and to be supernumerary to the establishment pending absorption, 20th February, 1927; Honorary Captain H. S. Roberts is appointed from the Australian Army Medical Corps Reserve and to be Captain (provisionally), 21st February, 1927.

The provisional appointment of Captain W. J. F. Douglas is terminated under the provisions of section 15 of the Defence Act, 28th February, 1927. *To be Captain (provisionally)*—Lieutenant W. J. F. Douglas, from the Reserve of Officers, 1st March, 1927.

*Australian Army Medical Corps Reserve.*

Honorary Captain A. H. Melville is transferred to the Australian Army Medical Corps Reserve, 3rd Military District, 1st March, 1927; Honorary Captain W. E. George is transferred to the Australian Army Medical Corps Reserve, 4th Military District, 1st March, 1927.

Honorary Captain J. O'Flynn is transferred from the Australian Army Medical Corps Reserve, 5th Military District, 20th March, 1927.

**Second Military District.**

*Australian Army Medical Corps.*

*To be Captain (provisionally).*—Horace John Foley, 10th February, 1927. *To be Lieutenant (provisionally)*—William Arnold Conolly, 11th February, 1927.

*To be Captains (provisionally)*—Colin Richard Cole and Cecil Walter Luscombe, 18th February, 1927; the resignation of Lieutenant (provisionally) N. J. Symington of his commission is accepted, 23rd February, 1927.

**Third Military District.**

*Australian Army Medical Corps.*

*To be Captain (provisionally) supernumerary to the establishment pending absorption*—William Langley Carrington, 7th February, 1927; the provisional appointment of Captain T. K. R. Allen is confirmed.

Captain T. K. R. Allen is transferred to the Australian Army Medical Corps, 1st Military District, 20th February, 1927.

*Australian Medical Corps Reserve.*

Major J. K. Richards is placed upon the Retired List with permission to retain his rank and wear the prescribed uniform, 25th February, 1927.

Honorary Captain A. H. Melville is transferred from the Australian Army Medical Corps Reserve, 1st Military District, 1st March, 1927.

Captain J. E. Butchart is placed on the Retired List, with permission to retain his rank and wear the prescribed uniform, 31st March, 1927; Honorary Major C. G. S. Godfrey is retired, under the provisions of Australian Military Regulation 152 (1), 1st April, 1927.

**Fourth Military District.**

*Australian Army Medical Corps.*

*To be Captain (provisionally)*—Leonard Charles Edward Lindon, 8th February, 1927;; Captain R. G. Burnard is transferred to the Australian Army Medical Corps Reserve, 7th February, 1927.

*To be Captain (provisionally)*—Lionel Deucalion Cowling, 23rd February, 1927.

*Australian Army Medical Corps Reserve.*

Honorary Captain W. E. George is transferred from the Australian Army Medical Corps Reserve, 1st Military District, 1st March, 1927.

Captain W. H. Jermyn is placed upon the Retired List, with permission to retain his rank and wear the prescribed uniform, 16th March, 1927.

*Award of the Colonial Auxiliary Forces Officers' Decoration.*

*Unattached List.*—Colonels M. H. Downey, D.S.O., and C. T. C. De Crespigny, D.S.O.

**Fifth Military District.**

*Australian Army Medical Corps.*

Major D. S. Mackenzie, D.S.O., is appointed from the Australian Army Medical Corps Reserve, 7th February, 1927; Major H. I. Carlile is transferred to the Australian Army Medical Corps Reserve, 4th February, 1927.

*Australian Army Medical Corps Reserve.*

*To be Honorary Captain*—Ernest John Tytler Thompson, M.C., 1st March, 1927; Honorary Captain J. O'Flynn is transferred to the Australian Army Medical Corps Reserve, 1st Military District, 20th March, 1927.

**Sixth Military District.**

*Australian Army Medical Corps.*

*To be Major*—Captain (Temporary Major) C. N. Atkins, 16th February, 1927.—(Ex. Min. No. 57.)

## Proceedings of the Australian Medical Boards.

### TASMANIA.

THE undermentioned have been registered, under the provisions of *The Medical Act* 1918, Tasmania, as duly qualified medical practitioners:

Kelly, Henry Patrick, M.B., B.S., 1926 (Univ. Melbourne), St. Mary's.

Whitworth, James Lister, M.B., B.S., 1924 (Univ. Melbourne), King Island.

## Books Received.

SOLUBLES OU INSOLUBLES, par le Docteur Henri Drouin; 1926. Paris: Editions Paul Martial. Crown 8vo., pp. 106, with illustrations. Price: 6 francs.

ACTA LEIDENSIA EDITA CURA ET SUMPTIBUS SCHOLÆ MEDICINÆ TROPICÆ; Volume I.; 1926. Lugduni: S. C. Van Doesburgh. Imperial 8vo., pp. 341, with illustrations.

THE PRACTICAL MEDICINE SERIES, COMPRISING EIGHT VOLUMES ON THE YEAR'S PROGRESS IN MEDICINE AND SURGERY. Under the General Editorial Charge of Charles L. Mix, A.M., M.D.; General Surgery, Edited by Evarts A. Graham, A.B., M.D.; 1926. Chicago: The Year Book Publishers. Crown 8vo., pp. 726, with illustrations. Price: $3.00.

WHEELER'S HANDBOOK OF MEDICINE, by William R. Jack, B.Sc., M.D., F.R.F.P.S.G.; Eighth Edition; 1927. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 645, with illustrations. Price: 12s. 6d. net.

BICENTENARY OF THE FACULTY OF MEDICINE, 1727-1926, UNIVERSITY OF EDINBURGH: RECORDS OF THE CELEBRATION; 1926. Edinburgh: James Thin. Imperial 8vo., pp. 131.

PRACTICAL NURSING FOR MALE NURSES IN THE R.A.M.C. AND OTHER FORCES, by Colonel E. M. Hassard, A.M.S. (Retd.) and A. R. Hassard; Second Edition; 1927. London: John Bale, Sons and Danielsson, Limited. Post 8vo., pp. 423. Price: 4s. 6d. net.

BARRIER CHARTS FOR HEALTH OFFICERS: A SYNOPSIS OF PREVENTIVE MEASURES AGAINST COMMUNICABLE DISEASE, IN FOUR TABLES, by S. H. Daukes, O.B.E., M.B., D.P.H., D.T.M. & H. (Wellcome Bureau of Scientific Research); 1927. London: Baillière, Tindall and Cox. Price: 2s. 6d. net.

ANATOMY OF THE WOOD RAT: COMPARATIVE ANATOMY OF THE SUBGENERA OF THE AMERICAN WOOD RAT (GENUS NEOTOMA), by A. Brazier Howell; 1926. Baltimore: The Williams and Wilkins Company. Royal 8vo., pp. 225, with illustrations. Price: $5.00 net.

THE MEANING OF DISEASE: AN INQUIRY IN THE FIELD OF MEDICAL PHILOSOPHY, by William A. White, A.M., M.D.; 1926. Baltimore: The Williams and Wilkins Company. Post 8vo., pp. 220. Price: $3.00 net.

REPORT OF THE DEPARTMENT OF HEALTH OF THE CITY OF CHICAGO FOR THE YEARS 1923, 1924 and 1925, Compiled by Herman N. Bundesen, M.D., Commissioner of Health; 1926; Chicago: James T. Igoe Company. Royal 8vo., pp. 858.

SYMBIONTICISM AND THE ORIGIN OF SPECIES, by Ivan E. Wallin, Sc.D.; 1927. Baltimore: The Williams and Wilkins Company. Royal 8vo., pp. 182. Price: $3 00 net

CLINICAL PHYSIOLOGY IN RELATION TO MODERN DIAGNOSIS AND TREATMENT: A TEXT-BOOK FOR PRACTITIONERS AND SENIOR STUDENTS OF MEDICINE, by Robert John Stewart McDowall, D.Sc., M.B., F.R.C.P. (Edinburgh), with an Introduction by W. D. Halliburton, LL.D., F.R.C.P., F.R.S.; 1927. London: Edward Arnold and Company. Royal 8vo., pp. 432. Price: 21s. net.

MEDICINE MONOGRAPHS: VOLUME IX: ACTIONS AND USES OF THE SALICYLATES AND CINCHOPHEN IN MEDICINE, by P. J. Hanzlik, M.D.; 1927. Baltimore: The Williams and Wilkins Company. Royal 8vo., pp. 213. Price: $3.50 net.

MOTHER AND UNBORN CHILD: A LITTLE BOOK OF INFORMATION AND ADVICE FOR THE PROSPECTIVE MOTHER, by Samuel Raynor Meaker; 1927. Baltimore: The Williams and Wilkins Company. Demy 8vo., pp. 218. Price: $2.50 net.

THE DE LAMAR LECTURES 1925-1926 OF THE SCHOOL OF HYGIENE AND PUBLIC HEALTH, JOHNS HOPKINS UNIVERSITY, Edited, with an Introduction by W. H. Howell; 1927. Baltimore: The Williams and Wilkins Company. Royal 8vo., pp. 226, with illustrations. Price: $5.00 net.

## Medical Appointments.

Dr. Henry Pearlman (B.M.A.) has been appointed Government Medical Officer at Sofala, New South Wales.

* * * *

Dr. W. A. Bridgeford (B.M.A.) has been appointed a Medical Referee for the purposes of *The Workers' Compensation Act*, 1912-1924, at Boulder, Western Australia.

* * * *

Dr. T. P. Byrne (B.M.A.) has been appointed a Medical Referee for the purposes of *The Workers' Compensation Act*, 1912-1924, at Kalgoorlie, Western Australia.

* * * *

Dr. John Patrick Hardie (B.M.A.) has been appointed Government Medical Officer at Bowraville, New South Wales.

* * * *

Dr. Ronald James Hunter (B.M.A.) has been appointed Government Medical Officer at Moree, New South Wales.

## Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xx.

COMMONWEALTH DEPARTMENT OF HEALTH: Medical Officer.

## Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

| BRANCH. | APPOINTMENTS. |
|---|---|
| NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney. | Australian Natives' Association.<br>Ashfield and District Friendly Societies' Dispensary.<br>Balmain United Friendly Societies' Dispensary.<br>Friendly Society Lodges at Casino.<br>Leichhardt and Petersham Dispensary.<br>Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney.<br>Marrickville United Friendly Societies' Dispensary.<br>North Sydney United Friendly Societies.<br>People's Prudential Benefit Society.<br>Phœnix Mutual Provident Society. |
| VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne. | All Institutes or Medical Dispensaries.<br>Australian Prudential Association Proprietary, Limited.<br>Mutual National Provident Club.<br>National Provident Association.<br>Hospital or other appointments outside Victoria. |
| QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane. | Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing.<br>Brisbane United Friendly Society Institute.<br>Stannary Hills Hospital. |
| SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide. | All Contract Practice Appointments in South Australia.<br>Booleroo Centre Medical Club. |
| WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth. | All Contract Practice Appointments in Western Australia.<br>Yarloop Hospital Fund. |
| NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington. | Friendly Society Lodges, Wellington, New Zealand. |

## Diary for the Month.

APRIL 26.—New South Wales Branch, B.M.A.: Medical Politics Committee.
APRIL 27.—Federal Committee of the B.M.A.: Australia.
APRIL 27.—Victorian Branch, B.M.A.: Council.
APRIL 28.—New South Wales Branch, B.M.A.: Branch.
APRIL 28.—South Australian Branch, B.M.A.: Branch.
MAY 3.—Tasmanian Branch, B.M.A.: Council.
MAY 4.—Victorian Branch, B.M.A.: Branch.
MAY 4.—Western Australian Branch, B.M.A.: Council.
MAY 5.—South Australian Branch, B.M.A.: Council.
MAY 6.—Queensland Branch, B.M.A.: Branch.
MAY 10.—Tasmanian Branch, B.M.A.: Branch.
MAY 10.—New South Wales Branch, B.M.A.: Ethics Committee.
MAY 12.—Victorian Branch, B.M.A.: Council.

## Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

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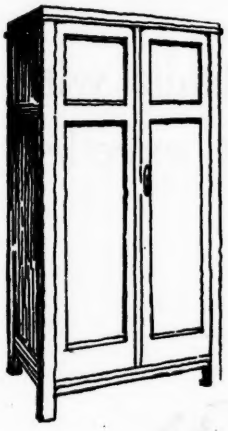
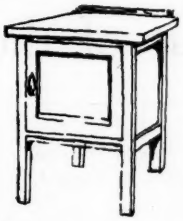
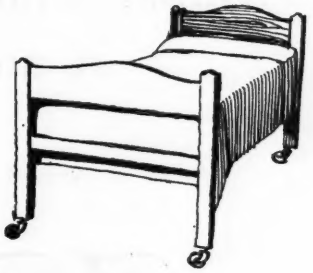


## The "Cobham" comprises:

3ft. WARDROBE, 20 inches deep, with two doors. Interior fitted with rod, hooks and mirror, 20in. x 12in.
2ft. 6in. DRESSING TABLE, with mirror 20in. x 16in. and two large drawers.
PEDESTAL CUPBOARD to match.
3ft. COMBINATION BEDSTEAD, fitted with iron frame and 3-ply wire mattress. Extra £5/10/-





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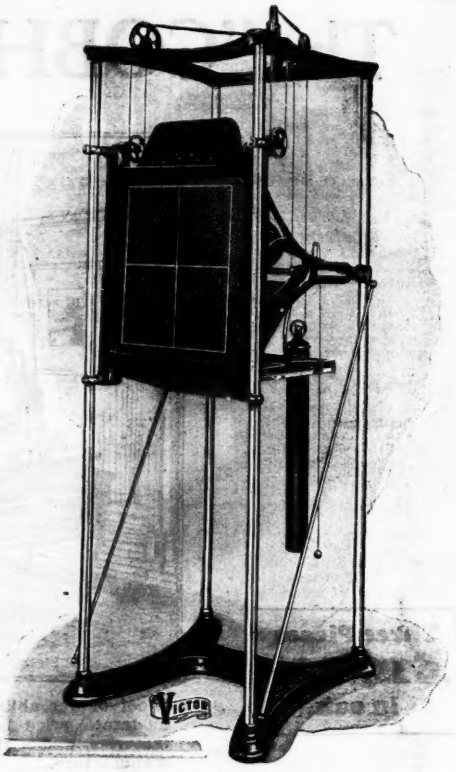


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